

# CLINICAL HEART DISEASE

BY SAMUEL A. LEVINE, M.D., F.A.C.P.

Assistant Professor of Medicine, Harvard Medical School; Physician,  
the Peter Bent Brigham Hospital, Boston; Consultant Cardiologist,  
Newton Hospital; Physician, New England Baptist Hospital, Boston

for  
Lucie

*Third Edition, Revised and Reset*

W. B. SAUNDERS COMPANY  
PHILADELPHIA AND LONDON





CONTROLLED BY

A

L578C



ALLAMA IQBAL LIBRARY



4699

Copyright, 1936 and 1940, by W. B. Saunders Company

Copyright, 1945, by W. B. Saunders Company

Copyright under the International Copyright Union

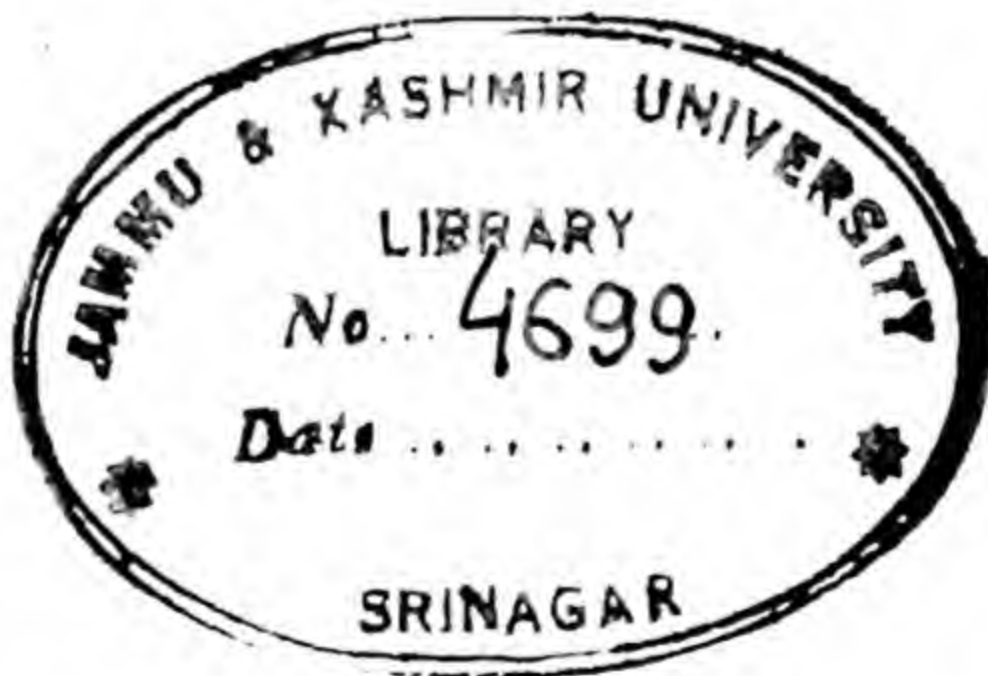
All Rights Reserved

This book is protected by copyright. No part of it may be duplicated or reproduced in any manner without written permission from the publisher

Reprinted June, 1945, October, 1945, July, 1946, January, 1947, September, 1947, September, 1948 and February, 1950

ST 01

R61



ST/S2

MADE IN U. S. A.

PRESS OF  
W. B. SAUNDERS COMPANY  
PHILADELPHIA

*In Gratitude  
To My Teachers*

HENRY A. CHRISTIAN  
ALFRED E. COHN  
JOSEPH H. PRATT

*Who first interested me in  
the study of heart disease*



## PREFACE TO THE THIRD EDITION

THE warm acceptance, by the medical profession, of the first two editions of this book warrants it being kept up to date. The book has not been changed in its general character. It continues to be a simple discussion of the common problems of heart disease, constantly bearing in mind the viewpoint of the general practitioner. For that reason, bibliographic references are deliberately omitted and some repetition occurs to avoid the turning of pages from one section to another. Additions and corrections in the text have been made as the viewpoint concerning the nature of the problem or its treatment has been changed. Many of the suggestions contained in the book reviews of the last edition have been incorporated. The reviews have been more than kind and complimentary, but I am particularly grateful for the constructive criticism they contained.

The discussion of such new developments as the surgical treatment of patent ductus arteriosus and the chemotherapy of subacute bacterial endocarditis has been amplified, particularly the use of penicillin. Brief reviews of subjects hitherto not included have been presented: *e.g.*, scleroderma heart, rupture of valves, and the heart in Addison's disease. Finally, two major additions have been made. Because of the increasing interest and importance of electrocardiography, and especially of the precordial lead, the discussion of this section has been elaborated. Many new electrocardiograms have been added, illustrating the more accurate methods of diagnosis now available by means of precordial electrocardiography. The growing interest in heart sounds and murmurs and their registration is responsible for a brief discussion of phonocardiography. A number of sound records have been inserted in the text in order that the reader might have a clearer idea of the significance of some auscultatory findings, such as gallop rhythm, changing quality of heart sounds and certain heart murmurs.

I wish to thank my wife, and my secretary Miss Gladys Kingman, for their assistance in preparation of the manuscript. I also wish to express my constant indebtedness to the house staff of the Peter Bent Brigham Hospital and to the fourth-year student clinical clerks of the Harvard Medical School. I am grateful for the stimulus derived from their perplexing questions concerning cardiac problems. The thoughtful co-operation of the publishers, W. B. Saunders Company, has lightened the task during these difficult days of the Second World War.

SAMUEL A. LEVINE

270 COMMONWEALTH AVENUE  
BOSTON, MASSACHUSETTS





## PREFACE TO THE FIRST EDITION

THE purpose of this book is to present in a simple form the important aspects of the diagnosis, prognosis and treatment of heart disease. It is meant to appeal to the general practitioner, and in so far as the information or the points of view that it contains are applicable at the bedside and available to any intelligent physician, just so far will it be useful. No attempt has been made to cover in detail the entire field of cardiovascular disease. Larger textbooks have appeared recently that have done this adequately. Nor does it contain any bibliographic references. For the most part opinions have been adopted that are shared by present-day authorities on the subject. When apparently unorthodox views are presented, I alone must bear the blame for error, if time proves these views to be incorrect. Where questions of opinion or speculations are involved, I have tried to draw the distinction between fact and surmise. This should not detract but rather add to the interest of a medical treatise, for unproved impressions often precede by years established dogma.

It cannot be said that the arrangement of the chapters follows any usual plan. Each chapter may be regarded as distinct in itself and as a brief treatise on that subject. The advantage of this is that they can be read independently. In fact, many of the chapters represent the essence of individual papers that I have published in the past twenty years with the help of various men working at the Peter Bent Brigham Hospital and reflect, therefore, the results of personal intensive study of the problems involved.

After the introductory chapter the various important types of heart disease are considered. When specific or peculiar modes of treatment arise, they are taken up as they come along, reserving the general subject of the treatment of congestive heart failure for the end. Special topics that concern the practitioner, which merit emphasis, are discussed separately. For example, because systolic murmurs are present in many forms of functional and organic heart disease and in fact even in normal individuals, a special chapter is devoted to their clinical significance. Similarly, acute cardiovascular emergencies arise under a variety of circumstances with and without organic heart disease and so, rather than discuss them in each chapter dealing with the respective type of heart disease, a special one is given over to this topic. In this way the reader can review all the types of cardiovascular emergencies for which a physician may be hurriedly called. Although the chapter on Clinical Electrocardiography was inserted at the very end, it may prove more useful to many to read it first.

Some repetition has seemed necessary and advisable in order to spare the reader from referring too frequently to one part of the book while



reading another. In discussing rheumatic heart disease, auricular fibrillation, for example, has to be considered. It also is taken up as a complication of acute coronary thrombosis, hyperthyroidism and other conditions. Nevertheless, when it is reviewed in the chapter on Clinical Electrocardiography, a brief summary is made of all the conditions in which auricular fibrillation is likely to occur. Apart from avoiding this necessity of constant reference to different chapters, such a method has an added advantage. It helps to give the reader two different modes of approach in medical diagnosis. One may start from a given known finding, like a certain irregularity of the heart or clubbing of the fingers, and review what the various causes may be, or begin with a known disease such as coronary thrombosis and predict what kind of complications may arise. Such repetitions, therefore, can only serve a useful purpose.

The hope is that this volume will prove practical. By this is meant that it will be easily understood and useful. It may seem that certain parts receive more than their proper share of space and emphasis. In general, points have been emphasized if they were simple, applicable at the bedside and of direct value to the patient. Little time needs to be spent in a discussion of those subjects or phases of medicine that are already well understood. A consultant with any extensive experience quickly finds out what is known and what is overlooked by the general practitioner. From this experience he can readily sense the emphasis that is needed in teaching.

If I may be permitted to digress a bit, I should like to express some views about our current methods of pedagogy in American Medical Schools. Inasmuch as the main purpose of our schools is to train men to go out into the active practice of medicine, we should keep constantly in mind that type of teaching that is practical and useful. The minority of our students, who are to become teachers and investigators, must and do receive post-graduate training in their respective fields. The initial undergraduate course, however, should be the same for all. This curriculum seems to lack a proper distribution of time with insufficient attention to that type of teaching that is most useful. For example, many hours are given to discussions concerning a subject like cancer of the pancreas which is entirely irremediable and too little to tumors of the spinal cord which are often completely curable. The former, of course, is more common, but the latter are more important because they are amenable to effective treatment. Granted that a medical student cannot be taught all we know about medicine in four or six years, it is more important, when he goes out into practice, that he should not overlook a case of spinal cord tumor with paralysis that has been diagnosed as amyotrophic lateral sclerosis or multiple sclerosis than to recognize a malignant growth of the tail of the pancreas. Likewise, it is much more important that a physician should be able to recognize the thyrocardiacs who are masked as heart patients and suffer invalidism (so readily pre-



ventable), than to be able to make an early diagnosis of subacute bacterial endocarditis. Until more is known about chronic arthritis and chronic nephritis it might be well to spend less time in our teaching of these subjects and more time with a rare condition like hyperparathyroidism, because the limb pains, renal insufficiency and other disabilities due to the latter can be readily eradicated or prevented by appropriate treatment. In a word, the first purpose in teaching is that the practicing physician should acquire that information which is directly helpful in the care of the patient. This does not mean that clinical investigations and laboratory research concerning the unsolved problems should be discontinued. A certain part of our profession must be constantly engaged in such effort.

Another aspect of medical education pertains to the simplification of medical diagnosis. In teaching hospitals and medical centers, elaborate laboratory facilities are readily available for diagnostic purposes. After an extensive *constructive differential diagnosis* has been built up, one possibility after another is eliminated by various tests. When that same house officer or student goes out into practice, he recalls the numerous possibilities involved in a given set of circumstances, but he no longer has the x-ray to rule out tuberculosis, a Wassermann test to eliminate syphilis and a blood culture to dismiss the diagnosis of septicemia. What simple clinical bedside methods remain to enable him to establish a temporary working diagnosis? In other words, how is he to disentangle the complicated differential diagnosis without putting the patient to great expense? This type of clinical teaching has been neglected, for there are simple methods that can be used in what might be called the *destructive differential diagnosis*, which the older or more experienced physicians have learned and which they are really practicing, consciously or unconsciously. A physician finds that a patient has a palpable spleen and fever. Among the various conditions to be considered is subacute bacterial endocarditis. He learned in his hospital training that a positive blood culture would establish the diagnosis, but that a negative one does not eliminate it. He has not been taught, however, that if there are no murmurs whatever, he can with fair assurance dismiss the diagnosis of subacute bacterial endocarditis. This finding he can obtain in one minute and with no expense to the patient. This merely illustrates one example, of which there are many, where simple methods enable one to rule out possible diagnoses. It would be desirable if our medical teachers paid more attention to this type of instruction.

A further difficulty in our teaching concerns the completeness or thoroughness of the examination. There are numberless tests and signs for various diseases. The practitioner cannot perform them all every time he sees a new patient. There is not enough time nor can the public afford the necessary expense. Therefore, we must not only teach these various procedures, but we should emphasize more than we do in our schools when these procedures should be carried out. For example, a systolic



thrill in the aortic area is an extremely important sign of aortic stenosis, and yet this sign is often overlooked. It can be missed when it is slight, because then it has to be detected by a special technic, *i.e.*, placing the palm over the upper sternum with the patient upright and holding a deep expiration. Physicians cannot and need not go through this procedure with all patients, but should be urged to do so only if there is also a fairly loud basal systolic murmur. Similarly, determining the visual fields is a specialized examination and will not be performed by most practitioners. However, it can be emphasized that, if there is some reason to suspect a pituitary tumor, a simple test for bitemporal hemianopsia can be performed in one minute by any physician. Moving a pencil on each side of the patient while he is looking forward and ascertaining when he begins to notice its movements will serve as a gross test of bitemporal hemianopsia. A further example is coarctation of the aorta. We must teach not only what the condition is, but under what circumstances it should be particularly sought. If a routine x-ray examination were made of the chest in all adult cases this diagnosis would not be overlooked. This is impracticable. We can emphasize that it needs to be thought of in all those who have hypertension, particularly in younger individuals, and if pulsations of the abdominal aorta or femoral arteries are diminished or absent, then further search for the evidence for or against this diagnosis should be made, even including the x-ray. In other words, teachers need to emphasize and simplify, more than has been done, those sets of circumstances in which special procedures either simple or complicated need to be carried out.

I want to take this opportunity to express my lasting gratitude to Dr. J. H. Pratt, who first excited in me an interest in heart disease while I was an undergraduate student. I also wish to thank Dr. A. E. Cohn of the Rockefeller Hospital for first teaching me the experimental method as it might be applied to the study of cardiac problems. All this would not have been sufficient if my chief, Dr. Henry A. Christian, had not afforded me every opportunity during the subsequent years for developing these interests. I well recall the early days in 1913 and 1914 when Dr. Christian first set up the electrocardiograph in the Peter Bent Brigham Hospital, having no one to turn to when this part or the other would not function. After giving me my earliest instruction concerning this new apparatus and the subject of electrocardiography, he set me off on my own. From then on I have remained constantly in debt to him for his stimulus and guidance in my work.

Much of the joy and stimulus has come from the undergraduate students, whose insatiable curiosity and perplexing questions must ever keep the teacher's interest alive, and from the many house officers and resident physicians of the Peter Bent Brigham Hospital who have helped me in these studies during the past fifteen years. We little realize the constant acquisition in knowledge that we experience from the casual and more spirited conversations with our intimate colleagues



and medical friends. This is one of the most characteristic and laudable aspects of our great profession. Among a host of such friends I cannot refrain from acknowledging my enduring gratitude to Dr. Frank N. Wilson of Ann Arbor, Dr. Tinsley R. Harrison of Nashville, Drs. Paul D. White and Soma Weiss of Boston, Dr. R. W. Scott of Cleveland and Sir Thomas Lewis of London. From all I have learned a great deal.

I also wish to thank Dr. F. Van Nuys of Weston, Massachusetts and Dr. W. D. Stroud of Philadelphia for their help in reviewing the manuscript and giving me the benefit of their criticism.

Considerable time and effort have been saved by the kindly services of Miss Bertha I. Barker, who has done all the technical work in electrocardiography at the Peter Bent Brigham Hospital these past twenty years. I also wish to acknowledge my obligations to the Oxford University Press for permitting me to use some of the figures in Chapter 21 that were previously published in their System of Medicine.

SAMUEL A. LEVINE

270 COMMONWEALTH AVENUE,  
BOSTON, MASSACHUSETTS



# CONTENTS

	PAGE
<i>Chapter 1</i>	
INTRODUCTORY CONSIDERATIONS . . . . .	1
✓ <i>Chapter 2</i>	
RHEUMATIC FEVER . . . . .	6
<i>Chapter 3</i>	
THE DEVELOPMENT OF RHEUMATIC HEART DISEASE: MITRAL VALVE DISEASE . . . . .	26
<i>Chapter 4</i>	
DISEASES OF THE AORTIC AND TRICUSPID VALVES . . . . .	43
<i>Chapter 5</i>	
DISEASES OF THE PERICARDIUM . . . . .	56
<i>Chapter 6</i>	
ANGINA PECTORIS AND CORONARY THROMBOSIS . . . . .	76
<i>Chapter 7</i>	
HYPERTENSIVE HEART DISEASE, ARTERIOSCLEROSIS, "CHRONIC MYO- CARDITIS," AND RARE FORMS OF HEART DISEASE . . . . .	126
<i>Chapter 8</i>	
THYROID HEART DISEASE . . . . .	142
<i>Chapter 9</i>	
SYPHILITIC HEART DISEASE . . . . .	152
<i>Chapter 10</i>	
BACTERIAL ENDOCARDITIS . . . . .	157
<i>Chapter 11</i>	
CONGENITAL HEART DISEASE . . . . .	170
<i>Chapter 12</i>	
FUNCTIONAL HEART DISEASE . . . . .	180
<i>Chapter 13</i>	
PAROXYSMAL RAPID HEART ACTION . . . . .	188
<i>Chapter 14</i>	
ACUTE CARDIOVASCULAR EMERGENCIES . . . . .	204
<i>Chapter 15</i>	
MEDICOLEGAL ASPECTS OF HEART DISEASE . . . . .	214

<i>Chapter 16</i>	PAGE
THE SIGNIFICANCE OF BRONCHIAL AND OTHER FACTORS IN THE PRODUCTION OF DYSPNEA . . . . .	219
<i>Chapter 17</i>	
THE CLINICAL SIGNIFICANCE OF THE SYSTOLIC MURMUR . . . . .	225
<i>Chapter 18</i>	
THE PATIENT WITH HEART DISEASE AS A SURGICAL OR OBSTETRICAL RISK . . . . .	234
<i>Chapter 19</i>	
FACTORS CONCERNING PROGNOSIS IN HEART DISEASE . . . . .	244
<i>Chapter 20</i>	
THE NATURE AND TREATMENT OF CONGESTIVE HEART FAILURE .	252
<i>Chapter 21</i>	
CLINICAL ELECTROCARDIOGRAPHY . . . . .	294
INDEX . . . . .	438



INTRODUCTORY CONSIDERATIONS

---

CONSIDERABLE knowledge has been gained since the turn of the century concerning the normal and abnormal changes that occur in the heart and the peripheral part of the circulation. One may ask whether or not this knowledge has improved the methods of treatment in actual practice. This question is often put with the inference that therapy has remained at a standstill and that the progress in our understanding, though interesting to physiologists and teachers and somewhat tedious to students, has merely made the subject-matter clearer and has put diagnosis on a more positive basis. Even if the latter alone were true and the diagnosis of heart conditions were now more accurate, a distinct advance of a practical nature would have been made. But as will be demonstrated, prognostication, although still difficult, has become much more definite and, in certain respects, treatment more efficacious. There are now patients suffering from certain heart affections who are treated effectively, for whom improvement may be expected and, in some instances, complete restoration of health obtained, whereas a generation ago similar conditions were entirely overlooked or improperly understood and utterly beyond any satisfactory therapy that was then available. It will become clear in subsequent chapters that even the ablest clinician of not so long ago was helpless before some of the problems that now respond dramatically to treatment.

These advances have resulted from the more careful clinical study of our patients, utilizing the common bedside methods of observation, the pathological study of autopsy material and the newer data that have come to us from the laboratory. With respect to the latter type of knowledge, we owe a great deal to the pioneer investigators, such as Mackenzie, Wenckebach, Einthoven and Lewis, who established modern cardiography on a scientific basis as a result of the introduction of the polygraph and the electrocardiograph. The use of basal metabolism determinations in clinical work has also materially helped our therapy. These and other laboratory procedures have now fortified our knowledge, so that, as a result of their use, we are successfully treating patients suffering from certain conditions that were previously regarded as hopeless. Reference is made at this point to the recent improvement of our knowledge and to the means by which the advance was obtained, to



combat the view that seems to be prevailing in the minds of many that laboratory methods have come to occupy too prominent a position in our medical study. An understanding of both the purely clinical and the more intricate laboratory aspects of disease are absolutely necessary for a proper approach to the diagnosis, prognosis and treatment of heart disease, and the slighting of one method or undue emphasis of the other, will diminish the accuracy and value of our work.

**Aims in the Treatment of Heart Disease.**—At the outset, the proper aims in the treatment of heart disease must be appreciated. Unlike other conditions in medicine, most sufferers from heart disease cannot be cured. The disease generally is a chronic one, and the purpose of intelligent care is the prolongation of life, the diminution of suffering and the increase of mental and physical efficiency of the patient. If that were all, it would be sufficient to warrant our serious effort and study, for sufferers from chronic conditions do and always will continue to seek medical advice for the above reasons alone, even when they know that a cure is beyond human attainment. If the difference between correct and incorrect advice given to a patient with early heart failure is a matter of two to five years of added life, then proper treatment renders much more aid than most of the unhappy sufferers of cancer obtain from the thousands of surgical operations that are performed for their relief. In addition, there is a comparatively small group of heart patients who present problems in which knowledge of the proper treatment saves life and effects complete restoration of health, whereas, with lack of that specific knowledge, fatalities occur. To be sure, such instances are uncommon, but to the few who succumb it is little comfort that these conditions are rare, and therefore we should be ready to render this invaluable service when the occasion arises.

There is a further aim to strive for in accurate study of heart disease which results purely from correct diagnosis. I have reference to distinguishing organic from functional heart disease. Many patients have been considered as suffering from structural heart disease because of certain signs or symptoms that we now know are benign. This results in great and unnecessary economic loss and unhappiness and frequently in the perpetuation, aggravation or actual production of a cardiac neurosis which might have been cured or prevented at the outset if the condition were understood. In other words, many sufferers from functional heart disease owe their disability to the inaccurate diagnosis made by some physician and to the effect produced by the fear and worry which such diagnosis engenders.

A final purpose that one hopes will be more important in the future than it is at present is the possible prevention or diminution in the incidence of heart disease that may follow a sound understanding of its problems. To be sure, we are now constantly hearing the cry of prevention from the lay public and the medical profession. It seems that with our limited available information, too much is being promised by



our medical brethren with regard to the prevention of heart disease. Although much is being said, little that is effective has as yet been accomplished, but the great importance of the subject warrants the tremendous agitation that is current.

**Normal Circulation.**— Before taking up the discussion of heart disease, it may be well to review briefly some of the simple events upon which a normal circulation depends. The main functions of the circulation are the distribution of oxygen and other nutrient and essential constituents, through the capillaries, to the tissues throughout the body, and the elimination of noxious products mainly through the lungs and kidneys. Let us at this point trace the different steps in the flow of blood within the body. The venous blood returning from the periphery enters the heart through the superior and inferior venae cavae into the right auricle. After an appropriate interval of diastolic filling of the auricle, during most of which time the tricuspid valve between the right auricle and ventricle is open, the auricle contracts and slightly less than one-fifth of a second later, the ventricle contracts. It must be appreciated that most of the blood, in fact about seven-eighths of it, goes from auricles to ventricles during diastole before the auricles contract, merely because of differences in pressure in the two chambers and the effect of gravity. Only a last bit of blood is ejected by auricular systole which gives the ventricle its final stretching before it contracts. When the right ventricle contracts, the tricuspid valve closes and the pulmonary valve opens. Blood is therefore sent through the pulmonary artery to the lung capillaries. There the essential change is the liberation of carbon dioxide and the absorption of oxygen in the alveoli of the lung, *i.e.*, the venous blood becomes arterial. The blood then returns by way of the pulmonary veins to the left auricle. The same movement of blood is going on in the left side of the heart, from left auricle through the mitral valve to the left ventricle, that was described above as taking place in the right side, only in the one case the blood is venous and in the other it is arterial. When the ventricles contract, the mitral valve closes, the aortic valve opens, and the arterial blood leaves the left ventricle through the aorta to enter the systemic circulation and to nourish the various organs of the body. The blood returns from the capillaries through the veins back to the heart to start the cycle all over. The flow of blood is essentially a dynamic phenomenon and results from differences of pressure in one part of the system as compared to another.

The above events recur with a rhythmic regularity under normal conditions at about the rate of seventy times a minute. The disturbances in this rhythm that occur in certain normal and abnormal states are taken up in Chapter 21, familiarity with which is essential to a precise understanding of the treatment of heart disease. These events produce two heart sounds that may well be described as lub-dub, or the first and second heart sounds. The first heart sound is essentially the result of the contraction of the ventricles and the closure of the mitral and tricuspid



valves, and the second sound is the result of the closure of the semilunar valves (the aortic and pulmonary). It has been maintained by some investigators, notably Dock, that the first sound is entirely valvular and that the contracting muscle itself produces no sound. The interval between the first and second heart sounds is systole and that between the second and first is diastole. The length of the former is approximately two-thirds of the latter. It will be seen later that in some cases, in order to avoid overlooking important findings in the heart that are to be heard by auscultation, it will be necessary for the physician to train himself to listen to one of the four features independently of the others. With a little training it is not difficult to dissociate from one's mind everything except the quality of the first or second heart sound. Likewise, certain murmurs that occur in systole or diastole will be heard only if the mind is concentrated for a given length of time on the interval between the first and second heart sounds or between the second and first sounds. I have emphasized this particular point because it has frequently happened that important diagnoses were overlooked as a result of aimless and more casual auscultation instead of concentrating attention upon a single element at a time while listening to the heart.

**Forms of Heart Disease.**—Before taking up any discussion of heart disease, it would be well to classify the general paths along which a heart may be diseased and the varying ways by which such disease may become manifest. A very common affection of the heart results from deformation of the valves. Such abnormalities may produce a regurgitation of blood through valves at a time when they should be closed or a constriction of the valves impeding the free flow of blood from one chamber to another when they should be wide open. The point of view I should like to emphasize at this time is that damage to valves is of great importance in undermining the efficiency of the circulation apart from the health of the heart muscle. To express this somewhat differently: if we assume the valves to be seriously injured at a time when the heart muscle is normal, progressive heart disease and failure of the circulation may yet take place because of the mechanical embarrassment that exists.

A second form of disorder develops from changes in the musculature of the heart entirely apart from the integrity of the valves. Here, as a result of alterations in the coronary blood vessels or of more diffuse damage to the heart muscle from toxins or certain poisonous substances circulating in the body, heart failure may result. Good examples of this are coronary thrombosis or diphtheritic heart disease. Furthermore, the heart muscle may fail functionally even when not significantly diseased, if there is marked hypertrophy. In this case the blood supply to the heart may be relatively insufficient for the thick muscle fibers. An entirely different form of heart disease occurs when bacteria start growing on the valves of the heart. Here the heart muscle may be normal and efficient and the mechanical embarrassment to the circulation as a result of the valvular deformations of trivial importance. The victim is never-



theless suffering from an affection of the heart which is almost always extremely grave. It must be clear, however, that in this condition the disease presents itself with the picture of infection and sepsis and not as circulatory failure, whereas in ordinary myocardial or valvular disease the patients are apt to complain of varying degrees of shortness of the breath or chest pain.

Disease of the pericardium of itself may embarrass the circulation either as the direct result of the inflammation that is present in acute pericarditis or by the mechanical impediment to the normal free movements of the heart that follows a pericardial effusion or pericardial adhesions. Much in the same way, extracardiac conditions may affect the heart such as thoracic tumors that produce pressure on the heart or great vessels or emphysema that produces increased pressure in the pulmonary circulation.

Another form of heart disease, which is comparatively rare, is that which results from congenital abnormalities. Here, as a rule, the musculature is essentially normal but the chambers of the heart, its valves or partitions, or the large blood vessels are improperly constructed. The result is either an impediment to the flow of blood in the normal manner or an admixture of venous and arterial blood because of defects in the septa that divide the right and left sides of the heart.

Apart from the foregoing structural changes that account for most forms of heart disease there are disturbances in the mechanism of the beat itself which present distinct problems in diagnosis and treatment. Such disturbances occur in normal people as well as in patients who also have structural disease of the heart. For example, a perfectly normal individual may suddenly develop a paroxysm of tachycardia under such circumstances that very disastrous results may ensue. Here the mere acceleration in rate enfeebles the circulation although there is no disease of the heart muscle or valves and no infection. A similar situation may develop in a patient who already has mitral stenosis. Then the embarrassment of the circulation may be more serious and develop more quickly. Such abnormalities in the mechanism of the heart may properly be regarded as functional and will be taken up in detail later.

There is a final condition which goes by a variety of terms that will also deserve our consideration. I have reference to functional heart disease and cardiac neurosis. These conditions were very prevalent during the First World War and had one or another of the following names: soldier's heart; effort syndrome; disorderly action of the heart (D. A. H.); neurocirculatory asthenia (N. C. A.); irritable heart; functional heart; or nervous heart.

The disability of which the patient will complain in one form of myocardial disease may be quite different from that in another. Dyspnea is generally the most prominent symptom of cardiac failure whether it occurs in a patient with valvular or myocardial disease. But when there is localized coronary artery disease producing angina pectoris, there may



be no dyspnea whatever and only chest pain. Likewise, the heart may be intoxicated as a result of hyperthyroidism without any dyspnea. Here the heart is actually hyperactive and the primary complaint may be palpitation. The inference from all this is that there can be no single method of testing the health of the heart. This explains why the various functional tests of the heart that have been devised, many of which are still being extensively used, have proved so unsatisfactory. Most of these tests utilize the effect of some effort on the heart rate, the blood pressure or on the production of dyspnea, in order to determine the health of an organ like the heart, which has so many different ways of expressing its abnormalities. It would be just as impossible to decide that the brain is normal by finding that the hearing or vision is not disturbed. As a further illustration of this difficulty, a patient may have a history of serious syncopal attacks with complete heart block (Adams-Stokes disease) and yet when put to a test of effort will show no dyspnea or pain whatever. The reason for this apparent anomaly is that the main disturbance in function in this particular instance is one of conduction of impulses, and the other functions of the heart are essentially normal. Functional tests in current use are for the most part tests of physical fitness and not of cardiac disease. Many individuals with normal hearts may manifest a poor response to effort and, contrariwise, those with definite well-compensated heart disease may show a normal or excellent response. The differences are really due to the degree of physical training or to variations of nervous stability in different individuals and do not measure the present or future status of the heart itself. The above will suffice to throw some light on the present status of functional tests of the heart and to indicate that the proper appraisal of problems in heart disease necessitates a complete survey of all factors that may have any possible bearing on the situation.



008.37619

## 2

## RHEUMATIC FEVER

THE term "acute rheumatic fever" has long been used in clinical medicine but is an extremely unsatisfactory one. The disease is often sub-acute or chronic rather than acute. There may be no "rheumatism" or pains, and fever may be very slight or absent. The term "rheumatic state" has come into use of late and has the advantage that it focuses attention on a "state" or peculiarity of the host, and yet a case of smoldering chorea need have no rheumatism. There is not even a con-



stant pathological finding, for the Aschoff nodule in the myocardium or elsewhere which is so characteristic is not always present. This is one instance in which it might have been advantageous to have a disease bear the name of some famous physician until its exact etiology were discovered, a custom in medicine that is often perplexing to students and practitioners. In this chapter rheumatic infection is meant not to include infectious arthritis, although in some cases the former seems closely allied to the latter.

Rheumatic fever is the most important infection that is directly related in a causative sense to the development of heart disease, particularly in younger people. With this is included chorea or St. Vitus's dance. The exact etiology of this disease is not known although the streptococcus is thought by many to be the cause. The evidence, however, is very conflicting and the question is best regarded for the present as unanswered! It is very likely that certain hemolytic streptococci play a role in this connection, as the disease so frequently follows in the wake of sore throats, tonsillitis or other streptococcic infections. The occurrence of epidemics of rheumatic fever in camps or institutions, where large groups are crowded together, following a widespread hemolytic streptococcus infection of the throat affords strong evidence in favor of the view that there is some causal relationship between the streptococcus organism and rheumatic fever. The exact relationship, however, is not well understood.

Much has been written of late about the allergic nature of rheumatism, comparing it to asthma, hay fever and urticaria, only that the sensitivity in the cases of the latter is to proteins and in the former to bacteria and their products, particularly streptococci. This conception has aided in the understanding of some of the manifestations of the disease. It can explain why joints may swell in response to a sore throat without the presence of bacteria in the joints. Under such circumstances certain tissues (*e.g.*, the skin or joints) may respond to infection or streptococci in distant parts of the body (*e.g.*, the tonsils, teeth or sinuses) because of local alterations in sensitivity. This point of view gives a different aspect to the idea of "focus of infection" in its relation to rheumatism.

At the outset it becomes very important to have a clear understanding of this particular infection and its various manifestations. The rheumatic infection often appears seven to fourteen days after some primary illness or upset. This primary cause generally is a streptococcus infection, like a sore throat, but may be an ordinary surgical operation, the injection of some foreign protein, a chilling or exposure, or the like. Scarlet fever is one of these trigger mechanisms that may start this series of events, but it cannot be regarded as a cause of rheumatic fever or rheumatic heart disease. On careful analysis it will be found that in only 1 to 2 per cent of the cases of scarlet fever are there cardiac complications. This percentage corresponds to that of rheumatic individuals in the general population. It will generally be true that the cardiac murmurs which follow scarlet



fever occur in that small number of patients who showed mild arthralgic symptoms about ten days after the onset of the disease. In other words, the scarlet fever infections uncover those individuals who are constitutionally rheumatic or vulnerable. It is not unlikely that if the scarlatinal infection had occurred at some other time when the particular host was not vulnerable it would not have resulted in a rheumatic bout.

We are all familiar with the typical attack of acute rheumatic fever in a child or a young adult who is suddenly afflicted with painful joints, which are tender, warm, swollen and red, the symptoms jumping from one joint to another in rapid succession. During such a condition there is a moderate fever and slight leukocytosis. This may last a few days, a few weeks, or even months. We are also familiar with the typical attack of chorea in which the child insidiously develops involuntary nervous muscular movements or twitchings of a peculiar character. Both these conditions may properly be regarded as different manifestations of the same rheumatic infection, for they frequently occur together; they attack the same type of individual; and in a sense produce the same disabilities in the heart. When either of these two conditions occurs in a typical form, it is very easily recognized. However, when the symptoms are slight, they are frequently overlooked both by the patient's family and by the physician, remain unrecognized, and lay the same foundation for the subsequent development of rheumatic heart disease. I have often seen children who have appeared somewhat nervous or fidgety, in whom it was extremely difficult to tell from the symptoms that chorea existed, and yet in whom there was sufficient evidence of a subsidiary nature, the importance of which will be discussed shortly, to make it certain that a true Sydenham chorea was present. The same has frequently been true of mild cases of rheumatic fever. Here the patient may only have vague aches and pains in the limbs, often called "growing pains," and yet, because of these same secondary manifestations of the rheumatic infection, the true character of the underlying disease became evident. We must, therefore, be ready to make the diagnosis of rheumatic infection in many atypical cases.

**Atypical Cases.**—The failure to recognize atypical cases of rheumatic infection accounts for the fact that in many instances outspoken valvular disease of the heart is seen in adults when no past history of rheumatic fever or chorea can be uncovered. In fact, if we take a condition like mitral stenosis, which I believe is due to only one disease, namely, rheumatic infection, in only about 50 per cent of the cases will there be a definite history of rheumatic fever or chorea. In the other 50 per cent, I believe that rheumatic infection occurred years previously, but presented itself in an atypical form, though with sufficiently characteristic features to be recognized if these unusual aspects of the rheumatic infection had been appreciated.

(We must look upon the rheumatic infection as a very protean disease, in many respects similar to syphilis. Apart from the more commonly



known organs that are involved, attention has been drawn to rheumatic lesions in the lungs, kidneys and blood vessels of other structures. It affects almost the entire body. A child has an attack of rheumatic fever at the age of nine, recovers satisfactorily, and then presents himself twenty years later with mitral stenosis. A young adult has a chancre at the age of twenty, and presents himself with aortic insufficiency twenty years later. Chorea may be regarded as the nervous manifestation of rheumatism, as meningitis is of syphilis. Both diseases have cutaneous symptoms, *i.e.*, the nodules and erythema multiforme of rheumatism as compared to the secondary rash or later tertiary syphilide. In both, the heart is frequently involved. The analogy may be carried further, for the joints and other organs are affected in both conditions. It is also true that the predominance of one type of symptom or another varies considerably from patient to patient in both diseases. In some syphilitics, the cutaneous features are very prominent; in others they are almost absent, and the central nervous system involvement is the outspoken lesion. Likewise, in some rheumatics, the element of arthritis may be entirely absent, and the nervous manifestation in the form of choreic movements may be the sole feature of the disease. In others, there may be neither joint nor nervous symptoms, and the disease will be confined entirely to an affection of the heart or of the skin. There are numerous instances in childhood in which the illness is characterized by gradual fatigue, lassitude, slight pallor, mild sweats, loss of appetite and weight, and a slight fever without any limb pains or chorea. Sometimes such a child will be considered as suffering from tuberculosis, possibly involving the hilus glands. The rheumatic nature of the disease is often overlooked and in fact could be suspected only by the most careful consideration of the secondary factors of rheumatism. In these cases it will frequently be found that the heart sounds are hyperactive or that a murmur over the precordium will be present either of which should direct one's attention to the possibility of rheumatism. One may be left in doubt as to the diagnosis only to see the child at some subsequent time go through a similar illness, this time associated with typical polyarthritides or chorea. We must, therefore, not confine the diagnosis of rheumatic infection to those patients suffering from typical polyarticular rheumatism or St. Vitus's dance.

The various manifestations of rheumatism are best looked upon as differences in the type of response on the part of the host rather than differences in strains or virulence of the infection. We all do not respond to the same insults in the same way. This applies to psychic, bacterial or mechanical trauma. One patient loses his entire fortune and then commits suicide, another after suffering a great financial loss takes to drink and a third grinds his teeth and starts all over again. Similarly one patient has a primary chancre and despite good treatment develops a stubborn skin syphilide. A second with little or no treatment has few or no skin lesions and only becomes aware of his plight when twenty years later



he has involvement of the central nervous system. A third has no involvement of the nervous system but develops an aortic aneurysm. Likewise two individuals cut their hands and blood flows. One faints at the sight of the blood and the other has enough presence of mind to put his handkerchief over the wound. These are differences in the response of the host and such differences are of extreme importance in rheumatic fever, for they explain the multiplicity of the symptomatology. It also must be remembered that the host changes during different periods of life so that the response in childhood may be different from the response in adult life or old age.

**Detecting Atypical Forms.**—Appreciating the fact that the rheumatic infection need not appear in its typical form as polyarthritis or chorea, what means have we of detecting the atypical forms of this disease? There are numerous clues that have frequently been invaluable in diagnosis that may be called subsidiary or secondary features of the disease. None of them is characteristic enough to be pathognomonic, for they are vague and may occur in many other diseases; but it is surprising how often, when considered *in toto*, they make up a distinct clinical picture. In the first place I have reference to *epistaxis*. It is well known that nosebleeds occur in many normal individuals and in a variety of diseases, but I know of no group of individuals who have repeated epistaxis as frequently as rheumatic children. I do not refer to the nosebleed that comes from trauma. The epistaxis here is spontaneous and may occur for some years before the outspoken attack of rheumatic fever takes place; it may occur during the active rheumatic infection while the child is sick in bed with fever and polyarthritis, or after he has recovered, feeling quite well and attending school. Whether → this is due to a peculiar vulnerability of the small blood vessels of the body in these individuals, or whether the actual cause of rheumatic fever which lurks in the body for many years produces specific pathologic changes in the mucous membrane of the nose, is not clear. It must be appreciated, however, that the rheumatic infection has a predilection for synovial membranes. It affects the endocardium, the pericardium, the pleura, the peritoneum, the synovial membranes of the joints, of the eye and possibly of the nose. At any rate, a history of repeated nosebleeds, together with other features, of doubtful significance in themselves, should make one strongly suspect that the patient is rheumatic.

Another symptom, although not quite as frequent as epistaxis, but one which may similarly be used in making a diagnosis of rheumatic infection, is *repeated vomiting spells*. The child may be ambulatory, attending school and suddenly have an attack of vomiting. This is generally painless and accompanied by only slight nausea or none at all. To be sure, vomiting is a frequent occurrence in many non-rheumatic children, but it has impressed me that recurrent attacks of vomiting are more common in this disease than can be accounted for as an accidental phenomenon. Sometimes, with or without this vomiting, there is pain



in the abdomen and tenderness. One can readily see from this brief description how a child might erroneously be operated on for acute appendicitis, because of the symptoms of nausea, vomiting, pain in the abdomen, slight tenderness, fever and leukocytosis. I have personally seen several instances in which this mistaken operation had been performed. It is in these unusual cases that one may be entirely dependent on other features of rheumatism to arrive at a proper diagnosis, such as hyperactive heart sounds, murmurs, family history, and nosebleeds.

**Familial Factor.**—Further peculiarities of rheumatism pertain to the family history and the constitutional type of the individual. There is now no doubt whatever that there is a strong familial factor in rheumatism. What is not clear is whether the high incidence of the rheumatic infection in members of the same family is due to a particular hereditary element or whether it is due to the fact that members of the same family are exposed to the same environmental influences. If the disease has a contagious or infectious element in it (and well-established epidemics of rheumatic fever have been reported), it would not be surprising that two or more children in the same household should have the same disease. Even if it is not contagious, but dependent upon factors like dampness, unhygienic surroundings, overcrowding, diet and the like, one would also expect to find an apparent familial instance of the disease. I am inclined to the opinion that apart from the infectiousness of the disease, there is a distinct hereditary predisposition. In one family I know of two sisters and a brother who live in different parts of New England, yet one or more of the children of each sibling has had rheumatic fever or chorea. In this same family the children have had one or another form of rheumatic infection and their parents and grandparents have shown marked evidence of degenerative vascular disease, namely, hypertension and angina pectoris. Here we have a striking example of familial vascular vulnerability, the children developing the infectious type of circulatory disease and the parents the degenerative form. I have seen this combination of rheumatism in children and angina pectoris in the parents too frequently for the relationship to be entirely accidental. The inference to be drawn at this point is, I believe, that there are families with vulnerable vascular systems and that this vulnerability is both to the infectious and the degenerative form of heart diseases.

**Constitutional Factor.**—Much attention has been given to constitutional or anthropologic features of the individual in relation to disease. I have been interested in this problem in so far as it bears upon three distinct clinical conditions, namely, pernicious anemia, angina pectoris, and rheumatism. There seems to be no doubt now that there is a type of individual with fair skin, blue, gray or light brown eyes, and fair hair (prone to early grayness), that more readily develops pernicious anemia. Highly pigmented individuals are only rarely affected by this disease. In a similar manner, there seems to be a certain type that frequently develops angina pectoris. I refer here to the well-set, stocky, strong man



who has always enjoyed good health. In this type the muscles seem to be hard, the skin tight, and the forearms well rounded rather than of flat configuration. There are also some striking characteristics in the rheumatic cases. It is surprising how many freckled and red haired children, frequently with hyperextensive fingers, are seen in our heart clinics. This is the type characterized in England as having the rheumatic diathesis. Although I have not any statistical control studies, I feel quite certain that the incidence of such individuals among patients with heart disease exceeds that in the population at large. Another finding of possibly less importance is the appearance of the sclerae. Many of the rheumatic children show pinkish coloration of the sclerae, which is due to an increase in the number of capillaries that come in from the periphery toward the iris. This peculiarity, however, may not be constitutional, but rather the result of infection. The foregoing constitutional considerations, although not as yet established on a firm scientific basis, cannot be lightly brushed aside. They seem to be important enough to deserve further study and have actually proved distinctly useful in diagnosis.

**Regional and Seasonal Distribution.**—Other peculiarities of rheumatism pertain to its regional and seasonal distribution. There is no longer any doubt that rheumatism, meaning by this term rheumatic fever and its allied conditions, is distinctly more common in certain parts of the world than in others. It is much more prevalent in New England, for example, than in the southern states. For some time this was not believed to be true, and the apparent difference was explained by the fact that in any statistical study the terminology was confusing. In the North the disease might be catalogued under the term "rheumatic fever," and in the South under some other name such as "infectious arthritis" or "polyarthritis"; but when it was found that the incidence of mitral stenosis at autopsy in a general hospital of a large southern city was about one tenth as great as in a similar hospital in Boston, one could not avoid the conclusion that rheumatism must vary a great deal in its frequency in different parts of the country; for no matter what the original infection was called in the two cities, if the disease were equally frequent, mitral stenosis, the common result of this disease, and a condition which is eventually fatal, would necessarily have been found with equal frequency in any large series of autopsies performed in the two places. This regional difference in the incidence of the disease is important entirely apart from its possible bearing on the nature of the malady, for where the disease is prevalent, it makes it imperative for the physician to suspect its presence even on slight or doubtful evidence.

The seasonal variations are also of some importance. Until the specific etiology is known, we shall have no explanation of many of the features and peculiarities of rheumatism. Among these are the variations in the prevalence of the disease during the different months of the year. The early spring is a particularly precarious time of the year, both for the development of new cases and for the recurrent attacks in old cases.



There is a practical inference in this observation, for if we have any measures of protection or prophylaxis that may be beneficial, it is during the months of February, March and April that these measures should be carried out most energetically.

**Cardiac Damage.**—Rheumatic disease is primarily important in so far as it affects the heart. The acute problem, whether it be polyarthritis or chorea, of course produces a disabling condition, but one from which recovery is almost always complete. It may last, however, a very long time. The painful joints or the active chorea may come and go over a period of years. This aspect of the disease is trying on the patient, his family and on the doctor, but they may all find comfort in the knowledge that eventually all this disappears. The most distressing feature of the condition is the great frequency of cardiac damage, even when the original infection is apparently mild. It is difficult to estimate how frequently the heart is involved, because one would have to define what is meant by heart damage. If all the means that are now available to detect abnormality of the heart are utilized in studying this question, it will appear that close to 100 per cent of the cases show some evidence of heart damage. To be sure, many of these changes are slight and transient; others, although permanent, may produce no bodily discomfort to the patient nor diminish his future usefulness. But it is fair to say that more than half subsequently suffer a more or less serious organic cardiac condition.

It must be clear from the foregoing remarks that whereas the rheumatic infection may show manifold symptoms, it may also appear in a singularly pure form. In one case there may be a great deal of polyarthritis; in a second there may be rheumatic nodules and vomiting and nosebleeds may occur; in another none of these symptoms will be present and only active chorea will be noted. The disease is the same in all instances, but the response of the body is different. What is much more important is that in a fourth case none of these symptoms may be manifest, but there may be an acute affection of the heart. Here again the cause is the same rheumatic infection.

It is quite conceivable and logical to regard the rheumatic infection as a great deal more prevalent than has been recognized. It can be compared to the situation that exists in relation to infantile paralysis. In this disease there is reason to believe that for every case in which paralysis occurs, there are many others in which the same infection took place but no paralysis developed. Likewise, may there not be numerous instances of rheumatic fever in which the rheumatism is either absent or very slight? There are probably frequent instances in which the child has a slight fever and sore throat, shows no appreciable arthritis, but develops a heart murmur. Such patients would be considered as developing valvular disease of the heart not due to rheumatic fever, but rather as a result of a simple sore throat or without any known etiologic cause. In other words, there are probably many mild infections, rheumatic in



nature, which affect the heart, but which go unrecognized and later go to form that large group of patients with organic valvular disease in whom no past history of rheumatic fever can be obtained.

**Clinical Features.**—Let us now consider briefly some of the clinical features that the patient presents who comes down with a rheumatic attack. The onset of the disease may be abrupt or insidious. It often follows a sore throat or an acute tonsillitis. The patient may, within a short time, complain of rather severe pains in one joint or another, show a moderate fever, and begin to perspire. It is characteristic of this disease for one joint to clear up rather quickly, and for another to become troublesome. In the fulminating case, the joints are extremely tender to the least motion, and there is swelling, redness and increased warmth of the affected parts. Sometimes these symptoms recede spontaneously, or as the result of salicylate therapy. In other cases, the arthritis remains refractory to treatment for a considerable period of time. There are great variations in the severity, duration and stubbornness of the joint manifestations. Occasionally, the entire illness seems to subside within a few days or a week, and the patient recovers. More frequently there is an amelioration of symptoms, but the infection keeps smoldering for weeks, months, or even years. During the acute stage of the disease, the patient is apt to develop some secondary anemia, and look pale. In almost all cases, the heart is accelerated out of proportion to the degree of fever. This is an important feature of the disease. There is hardly any other condition in which the heart rate continues as rapid, let us say, with a temperature of  $100^{\circ}$  to  $101^{\circ}$  F. for as long a time as in rheumatic fever. It is a common experience to see the heart rate continue around 120 for months with a fever of only one degree. Even if practically all the symptoms have disappeared and the patient feels fairly well, a slight fever and a rapid heart may persist. I have seen instances in which a temperature of about  $100^{\circ}$  F. and a heart rate of about 110 lasted for several years. During all this time the patients were feeling quite well, attending school, and undertaking ordinary activities. Finally, without any particular treatment, the slight fever and tachycardia gradually returned to normal. There is no better proof of the chronicity of this disease than such experiences. This also throws light on how the rheumatic infection may lurk within the body, smolder in a comparatively inactive way, and suddenly, without any evidence of a reinfection, flare up and new symptoms appear. Reactivation is a term that well describes this peculiarity.

At different times during the period of activity of the disease there is frequently pain in the precordium. This sometimes is fairly severe and troublesome, and may occur without any clinical evidence of either pericarditis or pleuritis. The heart sounds have a hyperactive quality. This pounding of the heart troubles the patient, and he complains of palpitation. The quality of the sounds resembles very much that heard in hyperthyroidism. It is always a matter of great importance to determine,



if possible, whether any part of the heart is being involved during the acute infection, and if so, to what extent. Similarly one should try to foretell whether certain suspicious evidences of damage are likely to indicate a permanent structural injury or not. In many cases, this is rather difficult, and at times impossible. A more detailed discussion of this question will be taken up in the following chapter.

**Diagnosis.**—The typical case of acute polyarthritis with involvement of one joint after another and complete subsidence of the previously affected part is easily recognized. The response of the fever and the pain to salicylate therapy is fairly characteristic but by no means pathognomonic. In some cases salicylates are not very effective and at times other types of fever or pains respond to this drug. In the atypical cases a complete knowledge of the subject will be necessary to identify the condition as rheumatic fever. The family and past history, the season of the year, the occurrence of epistaxis or vomiting, the feeling of listlessness and fatigue, the presence of sweats or anemia, the peculiar acceleration of the heart and the increased intensity of the sounds, the development of cardiac murmurs, the detection of a skin rash like erythema multiforme or erythema marginatum, the presence of small rheumatic nodules in the scalp, elbows, feet and spine or over ligaments are all important aids in diagnosis. The sedimentation rate of erythrocytes is increased but so it is in almost any infection and therefore it is not particularly helpful. It does aid in estimating whether or not the process once identified is still in the active stage, and with the presence of fever and leukocytosis may serve as a guide as to the length of bed rest. Furthermore, there are peculiar immunological reactions that occur with rheumatic fever. During the week or two following the initial streptococcus infection those persons who are rheumatic or who are prone to develop symptoms of rheumatic fever are likely to show a high titre of antistreptolysin in the blood. In fact, when such bodies do not appear, rheumatic symptoms are not likely to develop. It seems that some change or response in the host is necessary before the manifestations of rheumatic infection can take place.

Finally, there are changes in the electrocardiogram that are of considerable diagnostic importance. Many patients with rheumatic fever show an increased conduction time or P-R interval (see Chapter 21). Inasmuch as this is quite rare in other infections, its presence is presumptive evidence of rheumatism. Likewise alterations in the R-T complex of the electrocardiogram somewhat resembling those seen in coronary thrombosis are not infrequent in rheumatic fever. These changes, especially in the P-R interval, at times are the main or sole evidence upon which a proper diagnosis can be made.

Infectious arthritis may at times be confused with rheumatic fever. Very rarely the same patient may have both diseases at different times. Arthritis rarely involves the heart and rheumatic fever does not permanently cripple the joints. Lupus erythematosus disseminatus may closely



simulate rheumatic fever. The former is practically always fatal, is confined almost entirely to females during the years of menstruation, and there are apt to be characteristic skin lesions on the face. Both diseases are alike in that they may involve the endocardium, pericardium, lung, pleura, kidneys and other organs. At times tuberculosis, periarteritis nodosa, undulant fever, subacute bacterial endocarditis and other infectious diseases need to be considered in the differential diagnosis.

**Treatment.**—The treatment of acute rheumatic fever at present is entirely symptomatic. Numerous attempts at specific serum therapy have been made, especially on the part of those who have believed that a certain form of streptococcus was the true cause of the disease. It has seemed to me that whatever good, if any, that has been accomplished by this sort of treatment may be explained on the basis of a non-specific rather than any specific effect. Streptococcus vaccine therapy has been shown to diminish slightly the degree of arthritic pains in those cases prone to recurrences. Some form of salicylates, either the sodium salt or aspirin, should be used for the relief of pain apart from any effect upon the heart. It diminishes the amount of joint pains and the degree of fever. It is unlikely that the salicylates alter in any way the degree of cardiac damage. Some studies have shown that the heart may be favorably affected by this therapy, but it cannot be said that any great improvement in the cardiac condition is obtained thereby. It is customary to give sodium bicarbonate with the salicylates in about equal doses. Three to 8 grams of sodium salicylate in divided doses may be given per day by mouth. Even much larger doses at times are warranted. If the stomach is upset, either by the disease or by this therapy, the salicylates may be given by rectum. In this case the total amount per day is given in one dose by rectum in 50 to 100 c.c. of water. If uncomfortable ringing of the ears develops, the drug may be omitted for a day or two and treatment reinstituted with smaller doses. As the symptoms and the fever subside, the dose should be diminished. It has been the custom on the part of many clinicians to keep the patient on a maintenance dose of sodium salicylate or aspirin after the acute stage of the disease has subsided. This latter dose will vary with the size of the patient from about 0.3 gram to 1 or 2 grams a day.

Coburn has very recently reported striking results following intravenous injections of very large doses of sodium salicylate, *i.e.*, 12 to 24 grams in 2000 to 3000 c.c. of solution, daily, for a few days. He has devised a chemical method of measuring the concentration of salicylate in the blood. The aim of this treatment is to obtain a level of 400 gamma per 100 c.c. of blood. He has claimed that the joints improve promptly and the heart escapes injury. It is too early to be certain that this new treatment is as beneficial as the first report indicates. Furthermore, the possibility of salicylate poisoning must be considered.

During the active stage of the disease, the painful joints should be protected by proper support in the form of pillows, and frequently it is



necessary to place a hood around the feet and legs as even the weight of the bed covers may be distressing. Bandaging the tender joints after applying oil of wintergreen may give added comfort. Occasionally, the pains will be so distressing that sedatives such as codeine or even morphine may be advisable for short periods of time. These details in the care of the patient are of considerable importance, as they are supportive and help to spare his vitality, which is needed to combat an illness that frequently is chronic.

There is no particular problem in the care of the bowels except that it is advisable that the patient have one movement a day, with or without the aid of any cathartic. Inasmuch as rheumatic fever is frequently accompanied by a great deal of sweating, it may be necessary to change the patient's clothing frequently and to watch the condition of the skin. The heart itself, in the vast majority of cases, needs no specific medication, although in patients who have precordial pain an ice bag may be helpful. Although digitalis is often given because the heart is rapid, I have seen no evidence of any beneficial effects except in rare instances when simultaneously with the acute infection there is congestive heart failure as well. The matter of food is of considerable importance. The diet should be liberal and nutritious. One should welcome an actual gain in weight, for, in most cases, the patient is already undernourished. I know of no reason for limiting the diet in any way. Every attempt should be made to encourage extra nourishment. The diet should be adequate in vitamins, especially in fruit juices, all the more so because it is thought by some that deficiency in vitamin C is an important factor in the development of rheumatic fever. This relationship is open to some doubt.

It is always a matter of considerable moment to decide how long to keep the patient in bed. This, of course, will depend a good deal upon the severity of the illness and upon the extent to which the heart is involved. One would prefer to continue bed care until there are no symptoms and the temperature, pulse, white count, and sedimentation rate have been normal for at least one month. In many cases, in order to accomplish this, the patient will have to be bedridden for many months, particularly if one waited for the normal heart rate to be resumed, for, as has been mentioned above, tachycardia may continue even for years. When the disease persists and smolders, showing only slight evidence of activity, the practical problem becomes extremely difficult. To obtain the desired result, one might have to confine the patient to bed for one or more years. At some point in the course of the illness, the question of diminishing returns comes in. In the average public clinic patient, if such a drastic procedure is carried out in the attempt to obtain a slight and somewhat questionable advantage by means of a time-consuming and troublesome plan of treatment, the child frequently loses years of schooling. When recovery takes place, which might possibly have been accomplished without the loss of so much schooling, the child finds him-



self handicapped economically in later years of life. There is also to be considered the additional cost to the family of this prolonged medical care. When the advantages from prolonged bed rest are obvious, the decision is simple. Almost any sacrifice should be made if permanent and severe injury to the heart can be obviated. It is only when this advantage is either very slight or doubtful that these economical features enter directly into the decision. I often permit a child of humble means to continue at school after a previous trial of bed care, although there is a slight fever and a few joint pains, whereas if the economic status of the family permitted private nursing and tutoring I should advise the child to remain in bed. The importance of these considerations would be evident to any one who has charge of large numbers of clinic patients.

**Prophylaxis.**—This discussion would be incomplete without a word concerning prophylaxis. The intimate relationship between sore throat and tonsillitis and the development of rheumatism has been known for many years. Very frequently an acute attack of tonsillitis or sore throat initiates either the first attack of rheumatism or a recurrence. This brings up the whole question of a focus of infection as the underlying cause in the persistence of this chronic disease. The direct inference from the foregoing statements is that removal of tonsils would have a beneficial effect on the condition. There has been a great deal of discussion *pro and con* concerning this matter, and, as yet, the final answer has not been determined. At first it was hoped that tonsillectomy after the initial attack of rheumatism would actually diminish the number of recurrences that would take place and the degree of subsequent damage to the heart. If this is true, it certainly is so only to a slight extent, for we often see the disease persist unabated after the most careful tonsillectomy. After this disappointing experience, it was thought that once the disease had started, removing the tonsils was like closing the barn door after the horse had escaped. It was then argued that the infection had spread from the tonsils and was lurking elsewhere in the body. It would follow, therefore, if this premise were true, that by removing the tonsils in healthy children before the first infection took place, rheumatism would either be prevented or at least diminished in its incidence. This, of course, would require very elaborate and extensive statistical study. Such a study has been carried out in Rochester, New York, where many thousands of normal children with tonsils and without tonsils have been followed over many years. To date the results are only slightly indicative of beneficial effects so far as the diminution in the occurrence of rheumatism and rheumatic heart disease is concerned. Certainly, there are large numbers of children who have their first attack years after tonsillectomy. At present, it would be fair to state, however, that children who have had their tonsils removed are slightly less liable to have rheumatism in the future than those who have not had their tonsils removed.



There are other practical aspects to the whole question of tonsillectomy that merit our consideration. If this operation is to be done it is safer during childhood than in later years. Although post-tonsillectomy lung abscess is a rare complication, it occurs much more commonly in adults than in children. The same is true of subacute bacterial endocarditis which occasionally develops after tonsillectomy in adults with valvular disease, and almost never in children. Finally, the operation is a much simpler procedure in children, requiring only a day or so of hospitalization. These are additional reasons for early tonsillectomy in rheumatic children. When a tonsillectomy of election is being contemplated it is better to have it performed in the month of May or June than in August, September or October, because during the latter months poliomyelitis is more prevalent and there is a possibility of developing a severe post-tonsillectomy bulbar type of poliomyelitis that might otherwise have been avoided.

The discovery of sulfanilamide and related chemicals, which already have established their great value in combatting streptococcus infections, has led to their use as preventives of recurrent rheumatic fever. It is too early to judge their value, but at the present it seems that these preparations have no beneficial effect after the rheumatic bout has started, and also have been ineffective in forestalling a recrudescence if medication is started after the sore throat has developed. It has been reported, however, that by taking sulfanilamide in constant doses of 0.5 gram ( $7\frac{1}{2}$  grains) three or four times a day during the dangerous months (October to April) the incidence of recurrences of rheumatic activity has been definitely decreased. This study has shown that the number and reappearance of hemolytic streptococci in the throat of these children could be checked by such continuous medication. It is a subject that deserves our serious consideration. Patients receiving such prolonged medication need to be observed carefully, for a few will develop toxic reactions, particularly a secondary anemia, necessitating a cessation of the drug.

A factor that interests me a great deal from the point of view of prophylaxis, is the question of weight. It seems to me that the obese child rarely is stricken with an initial attack of rheumatic infection. This has been particularly true with regard to chorea, although many such children become obese after the disease has started if they progress favorably. One may contend that the apparent relationship between the rheumatic infection and the lowered state of nutrition is that the latter is the result of the former rather than the cause of it. It is difficult to offer scientific proof in this regard one way or another, and one would, therefore, have to depend for the present on general impressions. But as a result of observation of a great many rheumatic patients, I cannot avoid the conclusion that undernutrition is conducive to the development and the prolongation of this disease and that a diet particularly rich in proteins should be given to vulnerable children.



This discussion brings up the relation between normal or average weights and optimum weights. Doctors and parents often refer to standard weight tables when they consider the weight of children. We must appreciate that the normal figures given in these tables are average ones, derived from thousands of so-called "normal" individuals. These figures are, therefore, averages of good, bad, and indifferent weights. One should strive, not for average figures, but for optimum ones. The best weight at any one part of life may be quite different from the average. I feel that the best weight for children and young adults is distinctly above the average, and as we shall see later in discussing degenerative heart disease, the best weight for the second half of life is distinctly below the average. If this view is true, the practical inference follows that a deliberate attempt to keep infants and children overweight might actually diminish the ravages of rheumatic fever. Such attempts might be more particularly applicable when other features like a positive family history or peculiar constitutional characteristics indicate that the child is more than ordinarily susceptible to rheumatism. The same efforts in the dietary care should be carried out even after the first attack has occurred. Recurrences, I feel, are less apt to develop, or are less apt to be damaging, if those who once had rheumatism gain weight or actually become somewhat obese.

There are other measures of a preventive nature that deserve some attention. It is a frequent experience that an attack of rheumatism quickly<sup>o</sup> follows exposure to wetness and chilliness. I have seen instances in which the first attack came immediately after the patient was soaked through to the skin in the rain or was chilled from lying down on cold, damp ground. This should make us caution our patients to avoid getting their feet wet or their body chilled, and we should call these matters to the attention of the parents of these children, so that they may take appropriate prophylactic measures. It is a simple matter for the physician to tell the patient or his parents that he ought to avoid sore throats or catching cold. This is easier said than done. No doubt upper respiratory infections have an intimate bearing on or are often responsible for initial or subsequent attacks of rheumatism, but we have, as far as I know, no certain or specific measures for avoiding these infections, except the general ones that pertain to bodily hygiene. Mouth cleanliness and appropriate dental care should be carried out. Much is being said about the importance of the teeth in their relation to systemic disease, especially rheumatism and rheumatic heart disease. I cannot convince myself that I have ever seen an instance in which this causative relation existed as far as rheumatic heart disease is concerned. Teeth have a more important bearing on the development of subacute bacterial endocarditis than they have on rheumatic fever. Notwithstanding this doubt and skepticism as to the role of infected teeth, I do urge all these patients to keep their teeth in a satisfactory condition. Two other procedures, the value of which has not yet been demon-



strated but which deserve attention, are vaccinations against colds and x-ray treatment of the throat. The former, in the opinion of some authorities, will diminish the incidence of upper respiratory infections, and if this can be accomplished vaccination should prove of value. The latter has some theoretical justification for its use. We know that radiation can destroy adenoid tissue in the throat. It has been used as a substitute for tonsillectomy by some physicians. There are numerous miliary tonsils on the posterior pharyngeal wall that cannot be enucleated surgically, each one of which might be regarded as a possible focus of infection. I have, therefore, advised radiation of the throat in some cases in which sore throats have persisted after tonsillectomy. What good has been accomplished thereby in these cases is still uncertain. An upper respiratory infection with fever deserves more than usual consideration in a rheumatic individual. During an ordinary "cold," whereas many of us continue our daily work with the hope that it will pass by without any further ado, the risk is so great in an individual with a possible rheumatic infection that it is expedient that the individual should remain in bed until the illness is well over.

A final consideration in the matter of prevention concerns the question of climate. As has been mentioned earlier in this chapter, there are parts of the United States in which rheumatism is comparatively rare. The question arises whether we can utilize this factor in a prophylactic way. Will an individual who might come down with rheumatic fever while living in Boston, for example, avoid the disease if he spends his life in Florida, Southern California or in a city like New Orleans, where the disease is less common? There are no statistical studies to give the answer to this question. It is logical to think that the disease might be avoided in this way. But this has reference to patients who have as yet not suffered from the first attack. The matter is quite different when we consider what might happen to a patient who has already had a rheumatic attack and then goes to a region where the disease is less prevalent. In other words, will recurrences, after rheumatism has once affected the individual, be less frequent in one place than in another, or does the fact that the disease has once obtained a foothold make it independent of external surroundings? I have on occasion advised parents to move their families out of New England to warmer climates when one of the children persisted in having recurrences of rheumatism despite all therapeutic measures that we carried out. Although such instances have been too few in my experience to draw any definite conclusions therefrom, it seemed that good was accomplished when a change to a warmer climate was made. However, uprooting the whole household is extremely costly and drastic, and naturally can be undertaken only by a few families. It would be highly desirable if we could have more data in this regard, for, although costly, if effective, the move would be worthwhile. The whole question of treatment and prevention of rheumatism and its complications presents a most difficult and unsatisfactory situa-



tion. The solution, therefore, will have to await the discovery of the actual cause or the true nature of the disease.

From a sociological and public health point of view slow progress is being made and much more is to be hoped for. There is already statistical evidence that rheumatic fever and rheumatic heart disease are on the decline in the United States. Rheumatic fever is mainly a disease of urban populations and its incidence is directly related to factors such as crowding, dampness and poverty. As these conditions are improved rheumatic fever will be more satisfactorily controlled. In this regard there is reason to suspect that improved ventilation will help matters considerably as droplet infection appears to play some role in the spread of streptococcal infection.

**Prognosis.**—The ultimate prognosis of any single attack of rheumatism is most variable. Recurrences are numerous and, in fact, are the rule. In only a small number of instances does death occur with the acute stage of rheumatism and then it is as a result of a pancarditis, frequently associated with nephritis. In half the cases, at least, permanent heart damage results. It has been thought that the more frequent the attacks, the more likely there is to be a cardiac complication. This may need some qualification for in many cases there are repeated bouts without any further cardiac involvement. Some patients, in fact, suffer no permanent heart damage after several attacks. Thus, in some instances, we might be led to believe that the "all or none" law applies, *i.e.*, if the heart is to be attacked the damage will be done in the first attack and the degree of cardiac involvement will be as great in those who have one as in those having multiple attacks. The younger the individual at the time of the first attack, the more likely the heart will be involved. There are certain peculiarities in the outcome, depending on the original type of rheumatic infection, which at present remain unexplained. Although we rightly regard chorea and rheumatic fever as merely different manifestations of the same disease, yet there are qualitative differences in the type of heart damage that results from them.

When a child has chorea and no other manifestations of rheumatic infection (pure chorea) the heart is much more rarely involved than when rheumatic fever occurs with or without chorea. This applies to the events during the immediate few years after the infection. I do not believe, however, that chorea will prove to be as benign as it seems, if these same patients are seen years later; for I feel that many will still show valvular disease, in some because recurrent rheumatic infection will have taken place and in others even though there has been no subsequent infection. In other words, the child who had chorea at the age of eleven may show no evidence of heart disease at the age of fifteen and yet appear at the age of thirty or forty with mitral stenosis, never having suffered from any relevant illness in the meantime. I have not infrequently seen women over fifty with mitral stenosis who only then began to have symptoms of heart disease, who had had chorea in child-



hood and no subsequent illness. It is baffling to conceive of what was taking place in the mitral valve all those forty or fifty years between the original illness and the development of significant valvular disease. If chorea were as benign a disease as an early follow-up study would lead one to believe, there ought to be a considerable number of adults who give a past history of chorea in childhood but who show no evidence of heart disease. This has not been my experience.

A further peculiarity of chorea is the predilection for involvement of the mitral valve when there is a cardiac complication. Although the mitral valve is the most common one to be affected no matter what type of rheumatic infection may have been the cause, it is particularly so when the antecedent illness is chorea, for aortic involvement and pericarditis are rarely seen in those who only have had chorea.

These qualitative differences in the cardiac complications are rather difficult to explain if we accept the prevailing view that rheumatic fever and chorea are due to the same underlying cause, for one would then expect similar types of complications as far as the heart is concerned. There are two possible explanations that may have a bearing on this. In the first place, there may be different strains of organisms that cause rheumatism, one having a predilection for the nervous system and the other for the joints. The organism with a predilection for the nervous system may more easily affect the mitral valve than the pericardium or the aortic valve. One may draw an analogy between this state of affairs and syphilis. It used to be thought that there were strains of spirochetes that had a predilection for the central nervous system and others for the cutaneous system. The other possible explanation is that the virus is the same, but that the hosts differ. There are certain other factors that lend emphasis to this latter view. Chorea, for example, is more prevalent in the female than in the male, whereas the reverse is true with regard to rheumatic fever. Here one might say that sex alters the type of response to the same virus, the nervous system of the female being more vulnerable than that of the male. May there not be certain anatomical differences in the valves of the heart between those which develop mitral stenosis and those that do not or that develop aortic disease? Recent work has shown that in some individuals the heart valves contain blood vessels and in others they are absent, whereas some years ago heart valves were thought to be entirely devoid of blood vessels. May it not be true that whether mitral stenosis develops will depend upon the presence or the extent of the blood vessels in the mitral valve, and that those which are entirely or comparatively avascular escape? Such anatomical considerations may account for the discrepancies in the kind of cardiac complications that follow rheumatic infections.

### CHOREA

The general considerations as to the treatment of rheumatic fever apply with equal force to chorea. Just as the presenting complaint in



the one, *i.e.*, painful limbs, may vary in degree from practically no pain whatever to extreme, exquisite pain, likewise the nervousness may be manifest in all gradations from merely a "fidgety child" to the extreme "chorea insaniens." Occasionally there is marked weakness and paresis of an affected limb. I once saw a patient in whom the mistaken diagnosis of poliomyelitis was made. During active chorea there is apt to be no fever or only a very slight rise in temperature and the heart rate is generally slow or at least not as accelerated as during an active rheumatism. The treatment for active chorea is a prolonged period of rest in bed and general nursing care. Although various medicines are used and each physician may have his preference there is little evidence of a specific value that can be attached to any of them. Some prefer bromides or luminal, others use arsenic in the form of Fowler's solution, while a third group believe that sodium cacodylate intramuscularly is of value. At times the nervousness seems to be helped by daily warm tub baths.

→ (As I have watched the various methods used I have felt that isolation in a hospital with good nursing, bed care and forced feeding were the main factors in recovery.) When the chorea is of the extreme or maniacal type morphia may be indicated or even avertin by rectum (70 milligrams per kilo) may be given. Fever therapy has been advocated and found beneficial in shortening the duration of active chorea. The temperature of the body is raised to about 105° to 106° F. by means of a "hot box" and kept at that level for several hours. Such treatments are repeated once or twice at intervals of three to five days. In all but rare instances recovery takes place, for the cardiac complications during active chorea are hardly ever of any great immediate concern. The nervous twitchings may continue in a mild form with irregular exacerbations for several years.

There is one type of chorea that deserves special mention, *i.e.*, the chorea of pregnancy. Both rheumatic fever and chorea occur most frequently in the second decade of life, particularly during the ages of ten to thirteen. When once either form of the disease has appeared there is a marked tendency for subsequent attacks. In some instances the same manifestations return year after year while in others, a patient who first had chorea will later develop rheumatism or vice versa. After the period of full growth has been passed and the patients have reached the age of about eighteen years recurrences become much less frequent. From then on, more purely cardiac complications become manifest with repeated bouts of fever. Rheumatic pains and even outspoken acute polyarticular rheumatism, however, are not at all rare in the third and later decades. Occasionally even the first attack of rheumatic fever may take place during these latter decades. Curiously this is hardly ever true of chorea. I have not seen a single instance of chorea occurring for the first time in a patient past the second decade, except in the rare instances when it is associated with pregnancy. Even then there probably will be found a history of some manifestation of previous rheumatic infection. In other words, chorea does not occur in fully grown adults except in



association with pregnancy. (The great prevalence of chorea (and, for that matter, rheumatic fever) during the period of active growth of the child and the fact that chorea is never seen again except in association with pregnancy, brings to one's mind the possibility that the endocrine balance or the calcium metabolism is in some way related to the susceptibility to rheumatic infection.) For during both of these periods there is a great disturbance of the glands of internal secretion and an unusual demand on the calcium metabolism in the production of the long bones of the individual during growth and in the laying down of new bones in the fetus during pregnancy. ←

A final side-light on the nature of the rheumatic infections is the peculiar seasonal incidence mentioned above. The early spring seems to be the most prevalent time of the year both for first attacks and for recrudescences of the disease. This has interested me a great deal and particularly so in the light of work on animals at the Rockefeller Institute that showed seasonal variations in the susceptibility to experimental syphilis. It was found in this research that the weight of the various parenchymatous organs, particularly the glands of internal secretion, varied a great deal during different months of the year. February and March were found to be months during which animals were particularly susceptible to experimental syphilis. During these months, moreover, the relative weight of certain glands was especially low. It was also found that the calcium content of the blood varied in different months. May not similar factors be playing important roles in human susceptibility to rheumatic infections? In this connection the following experience is of interest. I once saw a young boy, eleven years old, who had just come down for the first time with an attack of chorea. It began during the month of February. I learned from the boy's mother that her brother (the patient's uncle) had had chorea when he was eleven years old and that he was first taken ill with it during the month of February. Thus, the same disease occurred in two members of the same family at the same age and during the same month. It makes one think that perhaps there is some inherent hereditary defect in metabolism associated with growth or with the glands of internal secretion that renders some individuals particularly susceptible to some noxious agent that is fairly generally prevalent. This conception that changes in the internal environment, especially the endocrine system of the host, may have a bearing on whether or not an individual will develop a rheumatic infection deserves more investigation than it has received, for it may suggest methods of prevention that so far have been wanting despite an enormous amount of bacteriological and immunological research. It at least gives added meaning to some of the vague phenomena, such as "growing pains" and "spring fever," that characterize this mysterious disease.

It is not unlikely that much valuable information will come to light in the near future concerning the relation of the endocrine glands to



many other diseases. Excision of the testes has already been found to have a favorable influence on cancer of the prostate. The fact that lupus erythematosus disseminatus is so overwhelmingly confined to women during their years of menstruations leads one to think that the ovaries play some role in this disease. The disappearance of a stubborn acne of the face (that failed to respond to many different types of treatment) directly after radiation of the ovaries is another example. The very low mortality of patients with untreated lobar pneumonia at the age of ten in contrast to the high mortality of those at the age of seventy may be related to differences in the endocrine system. Patients with thyrotoxicosis appear to have a much greater incidence of concomitant rheumatic heart disease and hypertension than other persons in the same environment. Women have very much less arteriosclerosis than do men and live several years longer on the average. Does this depend upon some function of the ovaries? One might cite other illustrations, all of which lend support to the view that the endocrine glands may have an important influence on vulnerability to infections as well as to the development of some non-infectious or metabolic diseases.



### 3

## THE DEVELOPMENT OF RHEUMATIC HEART DISEASE: MITRAL VALVE DISEASE

### ACUTE RHEUMATIC CARDITIS

THE development of rheumatic heart disease may best be studied by considering two aspects of the disease: first, what is taking place during the acute rheumatic infection and, second, the progressive changes that occur later, which go to make up the whole picture of chronic rheumatic valvular disease. During the acute rheumatic infection it is frequently difficult to tell whether or not the heart is being affected at all and whether certain changes that are noted indicate a transitory or a permanent cardiac damage. Often one has to leave the question open, and delay a final decision for some months or years. During this time the condition may possibly be called "potential heart disease." There are certain criteria, as we shall see, which afford quite conclusive evidence that the heart is involved. There are other changes that are common accompaniments of many febrile reactions, and, therefore, have not the same significance. The distinction between these two types of changes



is a most important one to appreciate in following a case of rheumatism.

While the active infection is going on, there is naturally some fever and acceleration in the heart rate. The tachycardia is frequently out of proportion to the degree of fever. This is quite typical of a rheumatic infection, but need not indicate that the heart is seriously damaged or will show structural changes after the infection is over. If the patient develops signs of circulatory insufficiency, such as marked dyspnea, congestion of the lungs and liver, or peripheral edema and the like, which are not common during the acute stage of the disease, we must assume that some serious injury is taking place in the heart. Even under these circumstances it is surprising how much improvement may eventually occur and how little may remain, after a long convalescence, to indicate any permanent organic heart lesion. When these symptoms are severe and a satisfactory recovery occurs, we cannot avoid the conclusion that there must have been an acute rheumatic myocarditis, in which healing took place without impairment of cardiac function.

**Systolic Heart Murmur.**—The most difficult feature to properly appraise during the acute infection is the development of a systolic heart murmur. We all are familiar with the fact that a very slight systolic murmur may be present during fever, whatever its origin, especially when the heart is rapid and hyperactive. A more detailed discussion of the significance of a systolic murmur is taken up in Chapter 17. However, the louder the systolic murmur that one hears during acute rheumatic fever, the more likely it is to be permanent and to indicate some organic lesion. This is more particularly true if a murmur of moderate intensity is present while the heart action is comparatively slow. As the heart slows, what might be regarded as a benign or functional murmur ought to diminish in intensity, for with the slowing the hyperdynamic element becomes less prominent. Loud systolic murmurs rarely disappear. Faint systolic murmurs may disappear; but if they persist they may be practically disregarded. The murmur of moderate intensity needs to be carefully followed and if it persists as the infection disappears, I believe that some injury, possibly minor in nature, has occurred although it need not incapacitate the patient in any way. It is clear from this discussion that we must pay some attention to the loudness of the murmur and to its persistence. When it is interpreted as indicating a structural damage, it generally points to involvement of the mitral valve, especially if its intensity is loudest in the apex region of the heart. This may even be true when the point of maximum intensity is at the base, although this is not so certain. We now know that the rheumatic infection may affect the wall of the aorta and this may add to the confusion of interpreting the causation of the basal systolic murmur.

**Disturbances in Conduction.**—A more certain indication that the heart is being affected during the acute disease is the finding of dis-



turbances in conduction in the mechanism of the heart beat. Not infrequently during the ordinary bedside examination, one detects an actual heart block, an occasional omission of an entire heart cycle or a dropping of the beat. This indicates an acute myocarditis, for the conduction apparatus of the heart lies within the musculature, and there must be some toxic process or some structural damage, like an Aschoff nodule affecting the auriculoventricular node or the bundle of His. Although it has recently been shown that this delayed conduction may disappear on full doses of atropine, indicating that it is vagal in origin, it is difficult to believe that such observations mean that the heart muscle has not been involved. Minor evidence of this disturbance in conduction which does not produce an actual blocking of beats, may be detected by the use of the polygraph or the electrocardiograph. Here it is found that the time it takes an impulse to go from auricle to ventricle is merely delayed, although the beats all reach the ventricle. The normal conduction time for impulses to go from auricles to ventricles is less than one fifth of a second. It is considered delayed if this upper limit is exceeded. The heart will then be perfectly regular and it might be impossible by bedside examination to discover this disorder. Occasionally, one may suspect it on detecting a gallop rhythm on auscultation, or on observing that the first heart sound is weak or fainter than it was formerly. If careful studies are made of the heart during the acute infection, changes in conduction are found to be quite common, and they are one of the best proofs that the heart muscle is being involved, at least temporarily. Fortunately, recovery from such damage is very likely to be complete, since, when the infection has passed, the conduction disturbances tend to disappear. Even in the rare instances, when some permanent defect remains as evidenced by the continued presence of a delay in the conduction time (P-R interval), the efficiency of the circulation may be perfectly normal.

**Electrocardiographic Changes.**—There are other changes of a qualitative nature, as shown by the electrocardiogram made during the acute process, that are fairly characteristic and that also indicate that the heart is being affected by the disease. This means of diagnosis is a more specialized one and can only be utilized by those clinicians who are carrying on electrocardiographic work. Suffice it for the present to bear this possibility in mind, for occasionally such minor changes in the form of the ventricular complex (see Chapter 21, Fig. 121) may be helpful from a diagnostic point of view, and may aid in determining whether or not a certain vague infection is rheumatic in origin. In fact, there will be instances in which the electrocardiographic changes will be the only evidence that the illness is of rheumatic origin. A girl of thirteen years who complained of listlessness, nausea and vomiting was seen by her physician. He found a slight fever ( $100^{\circ}$  F.), slight tenderness in the right lower quadrant and a leukocytosis of 14,000. The child was sent to the surgical service to be operated on for acute



appendicitis. A few hours after admission the temperature was only 99° F., and the white blood count was 12,000. The surgeon noted a faint abnormal sound over the precordium. There was very little evidence of any abnormal condition in the abdomen, but the electrocardiogram showed a P-R interval of 0.30 second. It seemed to me that the child had an atypical form of rheumatic fever of the abdominal type with a mild acute myocarditis, without any rheumatic pains. On rest in bed and salicylate therapy the patient recovered and the P-R interval returned to normal (see Chapter 21, Fig. 69). When the abdominal pain and tenderness are more marked than in this case, an unnecessary appendectomy is likely to be performed.

**Enlargement of the Heart.**—In the attempt to determine whether or not the heart has become affected during an acute rheumatic infection, the question of hypertrophy, or dilatation, of the heart is a matter of considerable importance. Although during the acute stages it cannot be said that a heart that is normal in size is not diseased, it may be safely accepted that if the size of the heart increases or is greater than normal, it is diseased. In fact, we may make the generalization that enlargement of the heart is always a definite sign of disease of that organ. There are important exceptions to this general rule. Appreciable dilatation without hypertrophy occurs with severe anemia, especially in children. This will naturally increase the percussion outlines and the x-ray silhouette of the heart. Such dilatation of the heart disappears completely when the blood returns to normal. Dilatation without increase in weight of the heart is also found occasionally in acute toxic states, particularly when the heart muscle is affected. It is, therefore, important to try to estimate the presence or absence of hypertrophy or dilatation. Ordinarily this is done by percussion and palpation. When it is possible to feel the apex impulse quite distinctly beyond the nipple line, it is fair to assume that the heart is enlarged. Frequently one is in doubt as a result of bedside examination whether the heart is enlarged or not, and further information in this regard may be obtained with the aid of the x-ray and the electrocardiogram. The use of the latter in estimating hypertrophy of the heart is subject to considerable limitation. The electrocardiograms may give indirect evidence of preponderant hypertrophy of one ventricle over another and occasionally may throw light on whether the auricles are dilated or hypertrophied. This type of data needs to be weighed most carefully, for there are numerous difficulties in interpretation. If it is decided by one means or another that the heart has become enlarged during the acute process, we must conclude that the heart has been affected.

**Other Evidences of Acute Rheumatic Carditis.**—While following any particular patient during an acute rheumatic attack, if enlargement of the heart has not been noted, if a systolic murmur is either absent or only slight and there are no obvious evidences of circulatory insufficiency, what other features are to be watched for? The development of a



*pericardial friction rub*, which may appear any time during the acute illness, will, of course, be definite evidence of cardiac involvement. Not only will the detection of a typical to and fro pericardial friction sound be proof of an acute pericarditis, but it may be assumed that in most such cases the heart muscle is also affected. Another condition that can develop during the acute process of the disease is an *aortic diastolic murmur*, heard in the aortic area and better still in the third left interspace near the sternum. This aortic diastolic murmur may be entirely absent on one day, appear as a faint blow a few days later and then be quite definite within a week or so. When it appears, it persists and is definitely indicative of an aortic regurgitation. A diastolic murmur of mitral origin is not to be expected to develop at this time, if we assume that the patient is now suffering from the first attack of rheumatism. It takes months or years for contraction and constriction of a valve to occur, for this is a chronic scar tissue process, whereas an insufficiency or regurgitation of a valve may take place within a few days. To produce the latter, it is only necessary for the valves to become slightly retracted or distorted by inflammatory reaction resulting in a slight incompetency. That same incompetent valve years later may contract as a result of scar tissue formation and then be stenosed. The important point is that in the acute infection we may look for insufficiency of valves, but if evidence of stenosis is detected, it is likely that the valve was previously injured and that we are not witnessing the first rheumatic infection but a recurrence.

Occasionally in children, during acute rheumatic carditis, a faint, short *diastolic murmur* may be heard at the apex resembling the murmur heard in mitral stenosis. Such murmurs must be interpreted cautiously for they may be due to dilatation of the heart or to some other mechanism and not to mitral stenosis, and they may disappear entirely. They are not apt to have the same rumbling quality that is found in older patients with mitral stenosis. In fact, errors of this type in the interpretation of the apical diastolic murmur as a sign of mitral stenosis are very rare in adults although not uncommon in children.

### MITRAL INSUFFICIENCY

Let us assume that the patient did not develop any of the definite signs of heart involvement during the acute stage of the illness and that if there were minor changes, such as a delayed conduction time or a slight change in the form of electrocardiogram, they were transitory. We assume that the patient had been in bed for some months with a smoldering fever, a rapid heart rate and vague fleeting pains in the limbs, and finally all these symptoms subsided. Let us also assume that he was left with a systolic murmur of moderate intensity heard best at the apex and that when he recovered he was symptom-free. In the course of time he returned to his ordinary activities either at school or at work and felt quite well. As we observe this patient during



the following years, let us trace what possible changes may take place. He may remain well, never have a return of rheumatic fever and always show a systolic murmur on examination. Such a patient would either be denied life insurance in his later years, or be considered an increased risk and receive a high rating in his insurance examination. Occasionally, the systolic murmur may gradually diminish in intensity. Rarely, it may disappear entirely. He may, therefore, live out his life as a normal individual and never be embarrassed by his heart. This, I think, is the exception, unless with recovery no murmurs whatever remained.

In a fair number of patients in whom all the findings on examination, including the systolic murmur, were considered benign, some years later, usually a decade or two, a fatal subacute bacterial endocarditis will develop. The remainder, either as a result of recurrent bouts of rheumatism and reinfections of the heart, or possibly because of the inherent nature of the original infection with its subsequent chronic progression and contraction without any recognized reinfection, will develop signs of mitral stenosis or aortic stenosis at some future time.

When this same patient whose progress we are following does not develop mitral stenosis but persists in manifesting a moderately loud systolic murmur while well and ambulatory, he may be considered as having organic mitral insufficiency. This is especially true if there is some evidence of dilatation of the left auricle or of general cardiac hypertrophy. It has been maintained by certain authors that organic mitral insufficiency does not exist or is extremely rare, and that in cases in which such diagnoses are made there is either no disease of the mitral valve or there is mitral stenosis. This point of view was a reaction against the previously prevailing belief that every patient with a systolic murmur had mitral insufficiency. We now know that in many such cases there is no structural disease of the mitral valve. They were often instances of nervous or hyperdynamic hearts with a benign systolic murmur or cases of myocardial failure of the degenerative type with a relative mitral insufficiency but with no endocarditis. Another group of patients at postmortem examination showed mitral stenosis. The conclusion was drawn, especially from the postmortem data, that organic mitral insufficiency did not exist without stenosis. The difficulty with this point of view is that we cannot deny the presence of a disease which is generally non-fatal by the use of autopsy data. It would be just as fallacious to maintain that acute tonsillitis and chickenpox are extremely rare because one almost never finds cases at autopsy. So it is with organic mitral insufficiency. This condition generally develops into mitral stenosis, which eventually is fatal, although for many years only an incompetency of the valve is present. As long as it is only a regurgitation, the patient is apt to be in good health. Even this is not invariably so for some patients die from heart failure showing a markedly dilated heart and mitral insufficiency without stenosis. Only occasionally have we an opportunity of examin-



ing the valve before stenosis develops, *e.g.*, if subacute bacterial endocarditis becomes superimposed on mitral insufficiency. Then we see that the past history of rheumatism and the moderately loud systolic murmur indicated a true rheumatic mitral endocarditis, producing a regurgitation but no stenosis of the valve; for the pathological examination will show both the old rheumatic and the recent bacterial lesions. The conclusion from the foregoing is that, although the diagnosis of organic mitral insufficiency should be made with caution, it is a condition that actually exists, especially in young rheumatic individuals.

### MITRAL STENOSIS

Let us now discuss the development of the signs of mitral stenosis. How soon the changes to be described will occur is a variable matter. Upon rare occasions they begin within one year, although generally many years elapse between the original infection and the development of definite evidence of mitral stenosis. During these years, the symptoms may be none or few, such as slight dyspnea and palpitation. If we examine the patient from year to year, little change may be detected until finally, as the first indication that the mitral valve is becoming stenosed, we commence to detect a snapping quality or accentuation of the first heart sound as heard at the apex. During these same years the pulmonary second sound may have become somewhat accentuated and reduplicated. As an aid in diagnosis, I have found the quality of the pulmonary second sound of distinctly less value than the quality of the first sound. In other words, an accentuation of the first sound is frequently the first suspicious evidence of early mitral stenosis. This, however, is not sufficient to enable one to make a definite diagnosis, for there are other conditions in which it occurs, such as hyperthyroidism, anemia, cardiac neurosis and some cases of hypertension.

When this snapping quality of the first heart sound is heard, it is well to listen carefully to the heart in order to hear the early development of presystolic or mid-diastolic murmurs. At this time no murmur whatever may be heard during diastole on ordinary examination, but if one listens directly after a brief effort or with the patient in the left lateral position, in some cases a presystolic murmur may thereby become audible which otherwise might be entirely overlooked. I have frequently brought out this very important physical sign which made a definite diagnosis of mitral stenosis possible when, under the ordinary examination, it was entirely inaudible. This typical rumble which is quite pathognomonic of mitral stenosis may also be uncovered by the use of the *amyl nitrite* test. Here the patient inhales amyl nitrite for a few seconds and one auscults carefully during the acceleration of the heart that follows the inhalation. In practice, it is sufficient to listen at the apex with the patient in the left lateral position or directly after the patient has had a brief period of exercise, *e.g.*, twenty-five hops. When the presystolic or mid-diastolic murmur is heard, no matter how



it is brought out, and the first sound is snapping in quality, the patient may be considered to have mitral stenosis.

At some later time it will be found that the same mid-diastolic or presystolic murmur will be audible even with the patient at rest or lying recumbent. It will be much better heard with the patient in the recumbent than in the upright position. Furthermore, the murmur may be quite sharply localized over a very small area in the region of the apex. As years go on and the degree of stenosis of the mitral valve increases, the presystolic or late diastolic murmur lengthens so that the time is reached when almost the entire diastolic pause is filled with the murmur. The early portion of this long rumbling murmur may have a diminuendo quality and the latter part a crescendo element, terminating in the accentuated first sound.

At this time we are not considering the development of generalized circulatory failure, which may come at any time during the patient's progress, but which generally does not occur until the mitral stenosis is well advanced. In the meantime the heart has been essentially regular in its rhythm, although there may have occurred occasional extrasystoles either of ventricular or auricular origin. The dominant rhythm, however, is likely to have been regular. Some time during the life of this patient who has already developed mitral stenosis, auricular fibrillation is apt to develop. This complication is very rare during the first decade, becomes more frequent the next decade and is most common about the ages of thirty-five or forty. Whereas the heart during all these previous years was regular, the beat now becomes tumultuous and grossly irregular. This change is sudden when it occurs, so that on one day a normal slow heart rhythm may have been present and on the following day it may be found to be very rapid and absolutely irregular. Not infrequently this auricular fibrillation is transient and recurs in the form of attacks. Each attack may last several hours and then disappear, the heart returning to its slow regular rate. Some weeks, months or years later the same phenomenon may be repeated. During the attack, the patient may suffer with a good deal of palpitation, dyspnea, and general nervous agitation. If the state of the reserve strength of the circulation is not great before the attack, the clinical condition of the patient may become quite serious. Edema of the lungs, cyanosis and orthopnea may develop. When an attack of auricular fibrillation with a rapid ventricular rate produces no evidence of cardiac failure, it denotes a satisfactory state of myocardial reserve. After a variable number of transitory attacks of auricular fibrillation, the condition is apt to become permanent. In fact, in most cases, once the heart becomes grossly irregular, it remains so indefinitely.

It is most important to be able to recognize this type of irregularity, first because it is extremely common, and second because much in the way of treatment can be done for it. Furthermore, a proper understanding of this phenomenon aids greatly in comprehending the changes



that take place when it occurs as evidenced in the physical examination of the heart. When auricular fibrillation develops, the auricles cease contracting, remain distended in diastole and show fine fibrillary twitchings throughout the musculature. The ventricles, on the other hand, begin to contract rapidly and very irregularly. In auricular fibrillation, there is an extremely large number of impulses traversing the auricles. The number is in the vicinity of four hundred or more. Only a small proportion of these impulses succeed in reaching the ventricles, the remainder being blocked at the junctional tissue. In other words, the conduction apparatus between the auricles and ventricles (the a-v node and bundle of His) is unable to transmit such a large number of irregular impulses from the auricles and only a third or so get through. The result is that the ventricles contract irregularly at the rate of about one hundred and twenty to one hundred and fifty. These contractions are grossly irregular. The pulse is also irregular, both in time and in force, and there is very apt to be an appreciable pulse deficit, *i.e.*, the pulse rate as counted at the wrist will be distinctly less than the heart rate at the apex.

It is not difficult to recognize this condition at the bedside examination using only the apparatus that we carry with us in general practice at all times. A rather simple and fairly satisfactory rule of thumb may be described as follows: given a patient who obviously has heart disease, shows a heart rate as counted at the apex of over one hundred and has a pulse rate that is distinctly less (a pulse deficit of ten or more), if the rhythm appears to be grossly irregular the condition is auricular fibrillation nine times out of ten. There are rare exceptions in which this rule would not apply. One would feel even more certain of the diagnosis of this arrhythmia under the above circumstances if it were known that the patient had mitral stenosis and a history of rheumatic fever. The triad of rheumatic fever, mitral stenosis and auricular fibrillation is so frequently found in the same individual that as a practical matter, knowing that two exist, the third should be suspected and be sought for.

There is one final point which may be helpful in detecting auricular fibrillation at bedside examination when there still remains some doubt in the diagnosis. Occasionally, numerous extrasystoles coming quite irregularly or from different foci of the heart may produce such a tumultuous rhythm, even with an appreciable pulse deficit, that it would be difficult to distinguish it from auricular fibrillation. One important distinction between the two conditions, however, will be noted on auscultation. In both conditions, quick beats and long pauses seem to be coming irregularly, resulting in sudden acceleration and sudden retardation of the heart beat. When the condition is due to extrasystoles, every time there is a long pause, it is a compensatory mechanism and therefore follows a previous quick beat. The same succession of long pauses after quick beats also obtains in auricular fibrillation. The phenomenon that distinguishes the two, however,



is the appearance of a long pause that is *not* preceded by a quick beat, for this occurs only in auricular fibrillation and not with extrasystoles. In other words, it is important to auscult carefully over the apex of the heart for a sufficient time to detect a sudden lengthening of the heart cycle and to recall whether the previous cycle was a short one or a long one. If a cycle of average length or one of longer duration is followed by a long pause, the condition is due to auricular fibrillation. Furthermore, the quality and intensity of the first heart sound vary much less with cycles of different length than is the case when the irregularity is due to extrasystoles. In addition to the foregoing observations, whenever it is possible to do so, an exercise test may help in diagnosis. When the rate accelerates in this manner, extrasystoles generally disappear and the irregularity of auricular fibrillation becomes more prominent.

Apart from the bedside method of diagnosis, auricular fibrillation is easily diagnosed by the use of graphic measures, such as the polygraph or the electrocardiograph. After a mechanical registration of the venous pulse is made, one obtains certain waves, two of which are primarily the result of the contraction of the right auricle and the left ventricle, respectively. The contraction of the right auricle sends an impulse upward through the vena cava and produces a slight wave in the jugular vein. The contraction of the left ventricle sends an impulse upward through the aorta and produces a wave in the carotid artery. The first is called the *a* wave and the second, the *c* wave. When auricular fibrillation is present, the auricles are no longer contracting, and, therefore, the *a* wave disappears. Likewise, in the electrocardiograms, the representative of auricular contraction (the P wave) disappears. In its place there may be found numerous fibrillary waves (*f* waves) running more or less throughout the heart cycle. These small waves represent the numerous fibrillary twitchings that are going on in the auricles in patients with this condition. In Chapter 21 these matters are taken up in greater detail. At this point, it may be stated that whereas cardiography gives the final proof of the diagnosis of auricular fibrillation, it is possible in the vast majority of cases to make accurate diagnoses by the use of simple bedside methods.

Let us now consider the effect of auricular fibrillation on the physical signs in a patient who has mitral stenosis. Before doing so it is necessary to review briefly the process involving the flow of blood from auricles to ventricles. As the ventricle begins to relax and dilate in diastole after the previous systole, blood flows from the left auricle into the left ventricle merely as a result of differences in pressure and gravity. The pressure in the ventricle starts from zero at a time when the left auricle has already received considerable blood from the lungs and has developed some pressure. In fact, normally about seven eighths of the blood flows from the left auricle to the left ventricle through the mitral valve by this mechanism. Only the last bit of blood is propelled



by the contraction of the auricle, which gives the ventricle its final stretching just before it contracts. It follows that the early and middle portions of the diastolic murmur of mitral stenosis are produced independently of auricular contraction, and it has been proved convincingly that only the presystolic portion is produced by auricular systole. Now, if auricular fibrillation develops in the patient we are considering, who had a murmur practically filling diastole, that portion of the murmur due to auricular systole will disappear because in auricular fibrillation the auricles cease contracting. In actual practice this is just what happens.

If, therefore, we examine the patient on one day when the heart is regular and find a murmur completely filling diastole, we should observe on the following day, if auricular fibrillation is present, that the presystolic interval is now clear and that the murmur extends only through the first portion of the diastole. It may be difficult or impossible during the early days after this change has occurred to appreciate the actual disappearance of the presystolic murmur. This is so because the heart rate is still rapid. When the rate is rapid, the diastolic pauses are short and they do not permit the silent portion of the diastolic interval to appear. Each first heart sound comes so soon after the previous second sound that, although the early portion of the diastolic murmur alone is present, it actually extends throughout the short diastole and gives one the impression that the presystolic murmur is still present. If, however, one listens carefully when the heart rate is slower, one finds that the diastolic murmur has remained unchanged. When the pauses are long enough and the following first heart sounds are sufficiently removed from the previous cycle, nothing whatever will be heard in presystole, *i.e.*, the presystolic murmur has disappeared with the development of auricular fibrillation. This discussion may seem complicated, but it is of some importance and deserves emphasis. The presystolic murmur by definition has its constant relationship with the following *first* heart sound, no matter how long the diastolic pause may be. If mitral stenosis exists and there is a normal mechanism, toward the end of the diastolic pause a rumble will be heard. The earlier and mid-diastolic portion of the murmur of mitral stenosis is not due to auricular systole and has its constant relationship with the previous *second* heart sound. It is that portion alone that persists when auricular fibrillation is present.

It is possible for auricular fibrillation to develop in a patient with mitral stenosis at any time, even when only the presystolic murmur had previously been present. The degree of stenosis may not be sufficient, or the dynamics may be such that the velocity of the flow of blood from left auricle to left ventricle is too slow to produce a rumble. One can readily see that if this occurred we might lose the only murmur in diastole that existed. The presystolic murmur would disappear entirely because the auricles were no longer contracting and there was



no other murmur in diastole. I have seen such cases in which the diagnoses were properly made and proved to be correct at postmortem examination. These, of course, are rare experiences. In one case, the patient was still thought to have mitral stenosis despite the absence of any murmur in diastole because on *x*-ray examination the left auricle was unusually prominent and the electrocardiograms showed right ventricular preponderance. These additional aids in the diagnosis of mitral stenosis will be taken up in greater detail below. In general, there are two types of cases of mitral stenosis when a murmur in diastole may be very faint or entirely inaudible. In the first type the stenosis is very slight and the state of the circulation very efficient. In the second type the stenosis is moderate or marked, but the degree of heart failure is considerable. The preceding discussion and particularly the experience just cited sufficiently emphasize the importance of the relation between auricular fibrillation and the murmurs of mitral stenosis.

I have paid considerable attention to auscultatory findings in mitral stenosis because I regard them to be of primary importance. One may gain the impression that a great deal of over-emphasis is being attached to the value of the stethoscope in this whole matter. To be sure, one obtains the most satisfactory appraisal of the degree of cardiac embarrassment from a brief inspection of the patient. The history of the symptoms of circulatory insufficiency, the degree of dyspnea, the distention of the cervical veins and the amount of passive congestion in the body give one much more important information concerning the state of the circulation than the determination of the presence or absence of a diastolic murmur. But in the vast majority of cases the final decision as to whether the patient has or has not mitral stenosis will depend upon auscultation. Symptoms and peripheral signs of congestion tell us whether or not a patient has *heart failure* but auscultation tells us whether or not there is *valvular disease*. Patients have valvular disease for many years before and for a longer time than they have heart failure. In fact, during the early years before cardiac insufficiency develops, without most careful auscultation it will frequently be impossible to tell whether the patient has organic heart disease or no heart disease.

There are other criteria that are useful in the diagnosis of mitral stenosis. The finding of a definite thrill in the apex region that is diastolic in time is quite reliable evidence of the diagnosis of mitral stenosis. When the heart rate is not rapid, the proper timing of this thrill is not difficult. When the rate is one hundred or over, it is almost impossible to distinguish the thrill that occurs in diastole from the vibration that is produced in a hyperactive heart. Such vibrations which are really systolic in time may be felt in patients with hyperthyroidism, in those who are in the acute stages of rheumatic infection and in those with certain nervous states. I have frequently observed instances in which such vibrations were misinterpreted and considered



to be diastolic thrills with the result that mistaken diagnoses of mitral stenosis were made. It has been my general experience that when the detection of the diastolic thrill could be made with certainty, auscultation would easily confirm the diagnosis, whereas in many cases in which the diagnosis of mitral stenosis was quite certain, thrills were entirely absent. The finding of the diastolic thrill, however, may have greater importance when there is a combined lesion of the mitral and aortic valves.

When hypertension is absent the volume of the pulse in mitral stenosis is customarily small (*pulsus parvus*). Cyanosis of the lips, cheeks and other portions of the face is common in mitral stenosis. Even when actual cyanosis is not present such patients are apt to have a rather florid countenance. This is in contrast to the pallor that typifies aortic disease. Mitral stenosis is also more common in the female than in the male sex. Another feature that characterizes mitral stenosis in contrast to disease of the aortic valve is its chronicity. Patients with mitral stenosis may continue to show cardiac failure over a great many years with repeated intervals of improvement. It is possible for such a patient to have congestive heart failure and be alive and in fair comfort five to ten years later. This does not occur with anything like the same frequency in aortic cases, for here the life expectancy is very short once decompensation develops. Hoarseness from pressure on the left main bronchus and dysphagia from pressure on the esophagus may result from enlargement of the left auricle. Rarely aphonia and paralysis of the left vocal cord occur from pressure of the dilated pulmonary artery on the recurrent laryngeal nerve. Pulmonary infarctions occur in mitral stenosis because of a tendency for the formation of mural thrombi within the cavities of the auricles, particularly in the auricular appendages when auricular fibrillation is present. From these thrombi, which may remain silent for many years, emboli may be dislodged and if they come from the right auricle they produce infarction of the lung, while if they come from the left auricle they produce peripheral emboli in the greater circulation with resultant hemiplegia, or other embolic complications. Pulmonary infarction is by no means always due to cardiac emboli, for it is a common finding in chronic congestion of the lungs no matter what the cause may be and can result from local thrombosis of the pulmonary vessels or from thrombosis of the veins of the pelvis or leg. *Hemoptysis* is also common in mitral stenosis apart from its occurrence as a result of pulmonary infarction. Such hemoptysis may be quite brisk, the patient raising a mouthful or more than a cupful of bright red blood. This may bring to one's mind the possibility of pulmonary tuberculosis or bronchiogenic carcinoma, both of which conditions need to be searched for in doubtful cases. It is striking how clear the lungs may be shortly after an attack of hemoptysis in mitral stenosis, as if the bleeding resulted from a rupture of a small dilated vessel and did not reflect a high degree of generalized passive con-



gestion. In some ways the hemoptysis resembles nosebleeds that occur in rheumatic individuals.

Considerable light has recently been thrown on this type of hemoptysis by Dock. It has been found by injection experiments that the bronchial veins can be enormously dilated in cases of mitral stenosis, whereas normally they are very small and difficult to visualize. They resemble miniature varices similar to those found in the esophagus in cases of cirrhosis of the liver. This mechanism does not apply to the cases in which bloody sputum is raised as a result of pulmonary infarction or in those in which there is acute pulmonary edema. In the former the sputum is blood stained or contains blood clots and in the latter pink, frothy sputum appears, while in the condition now being considered there is considerable pure blood in the absence of much obvious pulmonary congestion.

**x-Ray and Electrocardiography.**—There are times when the diagnosis of mitral stenosis is doubtful, even after the most careful bedside examination. There remain two means at our disposal which may further aid in this regard, *i.e.*, x-ray and electrocardiography. As a result of stenosis of the mitral valve, the pressure in the left auricle increases and this chamber dilates. This dilatation may be apparent on an x-ray film of the heart exposed in the ordinary manner. A bulge may be seen in the left upper border just below the pulmonary artery. Sometimes it is quite prominent. Occasionally it is difficult to distinguish this, except by fluoroscopic examination, from the dilatation of the pulmonary artery that occurs with patent ductus arteriosus or other conditions. Furthermore, the left auricle may extend with undue prominence posteriorly and bulge into the posterior mediastinal space. On rare occasions the left auricle will be so dilated that it will actually extend across the midline and form the right upper border of the heart. The large left auricle may produce a definite angulation of the esophagus and push it to the right. This can be observed on fluoroscopic examination or on a roentgenogram taken while a barium meal is being swallowed. Even the left primary bronchus may be seen on x-ray examination to be raised upward or constricted by a dilated left auricle. These observations may sometimes be extremely helpful in distinguishing mitral stenosis from congenital heart disease. Finally with improved x-ray technique, it has been possible to see calcification of the mitral valve on fluoroscopic examination and less frequently on the flat x-ray plate. When this is properly done it always means mitral stenosis and it can be distinguished from calcification of the annulus fibrosus which usually causes no disturbance in the function of the heart and is an unimportant finding. The detection of calcification of valves has already proved of great aid in diagnosis, especially when combined lesions are present.

The electrocardiogram occasionally gives helpful indirect evidence of mitral stenosis. As a result of hypertrophy and dilatation of the auricles in this condition, the auricular complex (P wave) may develop



a peculiar form, become unduly large, prominent, notched and have a flat top (see Chapter 21, Figs. 98, 99). When these changes are marked they are almost invariably indicative of mitral stenosis. A further change that results from mitral stenosis is hypertrophy of the right ventricle. The pulmonary pressure is increased and with it a compensatory hypertrophy and dilatation of the right ventricle take place. Inasmuch as the electrocardiograms give some measure of preponderant hypertrophy of one ventricle over another, in mitral stenosis they may show certain alterations from the normal. These changes consist of prominent downward deflection of the initial ventricular complex in Lead I and a prominent upward deflection in Lead III. Such curves, however, must be interpreted with great caution for not infrequently they may be obtained in subjects with normal hearts, in those with emphysema or in patients with certain forms of congenital heart disease. Electrocardiography in general has not been very valuable as an aid in valvular diagnosis, but occasionally this evidence, coupled with other data, which of themselves might not have been sufficient, enables one to make a proper diagnosis.

**Common Signs of Heart Failure.**—Throughout this discussion nothing has been said about the common signs of heart failure that may be found in a patient with mitral stenosis, such as engorged liver and cervical veins, generalized edema, enlargement of the heart, and the like. These are not particularly characteristic of mitral stenosis although it must be stated that enlargement of the heart in mitral stenosis is more apt to be transverse, especially to the right, than it is in other forms of heart disease. The signs of heart failure associated with mitral stenosis will be taken up in detail when discussing the problem of general cardiac failure.

Little has been said about the presence of a systolic murmur at the apex in mitral stenosis. This, of course, is a very common finding but does not indicate that the mitral valve is stenosed. It means that there is a concomitant mitral insufficiency or it has the questionable significance that accompanies systolic murmurs in general. It has been generally taught that mitral stenosis is almost always associated with mitral insufficiency. Whether or not regurgitation of blood actually takes place in many of these cases is difficult to ascertain. The only physical evidence of this would be the presence of an apical systolic murmur. Now, it is not sufficiently appreciated that, in a large number of cases of clearcut mitral stenosis, no systolic murmur whatever can be heard. The same may be said of stenosis of the aortic valve. I am of the opinion that mitral stenosis without any insufficiency is very common, and that it often develops in patients who previously never had a systolic murmur.

It also must be understood that mitral stenosis is not always the sole valvular lesion of the heart. There may be an additional involvement of the aortic or more rarely of the tricuspid valve. The diagnosis



of combined lesions is no simple matter, but in so far as it can be determined, it will depend upon the proper utilization of the criteria for each individual lesion with some regard for the effect of one upon the other.

**Mitral Stenosis and Blood Pressure.**—This discussion would not be complete without some mention of the relation between mitral stenosis and the blood pressure. The old term "pulsus parvus" reflects the general impression that the pulse is small and the blood pressure is low in this condition. In a study of almost 800 cases of mitral stenosis, it was found that when the disease was present in young people, the blood pressure tended to be lower than that of the average population of that age group. But in patients with mitral stenosis who were in the higher decades of life the average blood pressure increased decidedly until it reached 180 systolic and 95 diastolic for patients sixty to sixty-nine years of age. This I feel cannot be an accidental finding, for certainly no group of patients suffering from some unrelated condition, such as cancer of the breast or ulcer of the stomach, has hypertension as a general rule. It is hard to believe that mitral stenosis itself accounts for the hypertension, for when present in young persons, the pressure is even lower than normal. Yet how are we to explain the frequent association of mitral stenosis and hypertension?

One may at the outset maintain that the mitral stenosis in these older hypertensive patients is not rheumatic in origin and that it is due to sclerotic changes in the mitral ring, similar to the peripheral arterial sclerosis with which the hypertension is associated. One might object to the accuracy of the diagnosis of mitral stenosis in these elderly hypertensive persons on the ground that the presystolic gallop rhythm which is common in hypertension might be mistaken for the presystolic murmur of mitral stenosis. In answer to the latter it may be said that the diagnosis in a sufficient number of these elderly patients has been confirmed at autopsy. As far as the former criticism is concerned, apart from the general view that I have expressed before, in which it was stated that mitral stenosis is probably due to no other cause than rheumatism, it was found that 50 per cent of these hypertensive patients had a past history of rheumatic fever or chorea, which percentage corresponds fairly closely to that of a rheumatic history in any group of patients having mitral stenosis. Inasmuch as mitral stenosis *per se* does not produce hypertension, it is possible that the underlying chronic rheumatic infection insidiously produces arterial changes that terminate in hypertension. Another possibility is that a long standing disease process in the heart by some reflex mechanism can initiate hypertension. Furthermore, the great frequency of hypertension in the older patients with mitral stenosis may be due to the fact that the elevation of the blood pressure enabled them to survive and reach the later decades of life. A more likely explanation, it seems, is that mitral stenosis and hypertension are commonly associated



because the underlying rheumatic infection has a predilection for patients who have a vascular vulnerability. The disease has a tendency to select certain types of people and this so-called "vascular" type is vulnerable both to the infectious and to the degenerative form of heart disease. The frequent association of rheumatic heart disease and angina pectoris in the same family is additional evidence in favor of this conception.

There is also some clinical and statistical evidence to indicate that the hypertension developing in patients with mitral stenosis need not produce any deleterious effect, but on the contrary may be actually helpful. Although it may add certain symptoms as a result of the hypertension itself, when these are not severe, there is reason to believe that it delays the progress of mitral stenosis. It is very curious that so many patients with mitral stenosis over fifty years of age have hypertension in addition. I believe that the hypertension prolongs their lives and enables them to reach the later decades, for the great majority of patients with mitral stenosis without hypertension die before they reach the age of fifty. One may explain the beneficial effect of hypertension by supposing that as a result the cavity of the left ventricle tends to remain large and somewhat dilated and that this dilatation tends to counteract or delay the antagonistic, progressive contraction that is going on in the mitral ring as a result of the rheumatic mitral stenosis. Another possible factor is that mitral stenosis is a burden on the right side of the heart and hypertension embarrasses the left side of the heart. In this way the two burdens are equalized and if congestive failure is due to an imbalance of the two ventricles, as is believed by many authorities, the effect may be beneficial. Expressed in the form of a lay parable: if one is to have a soft or flat tire on a motor car it would be of advantage to have the one on the other side in a similar condition. I have frequently seen elderly patients with mitral stenosis and hypertension continue to do fairly well many years after it was expected that they would die. It therefore appears to be a somewhat favorable sign to find an increasing blood pressure in cases of mitral stenosis.



## DISEASES OF THE AORTIC AND TRICUSPID VALVES

THERE are certain striking differences between diseases of the aortic and the mitral valves. Aortic valve disease is more common in males than in females, and the reverse is true for mitral disease. Pain in the chest of the type that is characteristic of angina pectoris is frequent in aortic disease and is rare in mitral disease. Pallor is more typical in the former, whereas plethora or at least a florid countenance is more typical in the latter. Patients with aortic valve disease, although frequently complaining of chest pain and palpitation, have much less dyspnea as compared with those having mitral disease. They generally also have better bodily strength and remain ambulatory and able to do work for a longer time with outspoken evidence of valve disease; but when general circulatory insufficiency does develop, with dyspnea, congestion of the lungs and liver, or peripheral edema, they have not the recuperative power that is commonly seen in patients with mitral stenosis. In other words, when decompensation is once developed in an aortic case, the outlook is rather grave. The average length of life after such an occurrence is not apt to be more than two or three years. In mitral cases, on the other hand, frequently several attacks of decompensation occur, each followed by a partial restoration of circulatory efficiency. The patients are thereby enabled to continue for many years on restricted activities. Pulmonary infarctions are less common in aortic than in mitral cases. This may be partly but not entirely accounted for by the fact that auricular fibrillation is extremely rare in disease of the aortic valve and very common in mitral stenosis. Disturbances in conduction, such as heart block and bundle branch block, are much more common in aortic than in mitral disease. The same is also true of attacks of syncope and sudden death. A final difference that has impressed me in recent years is that subacute bacterial endocarditis is more common in aortic disease than in mitral stenosis. This does not mean that the mitral valve is less frequently involved in subacute bacterial endocarditis for in most cases when the mitral valve is involved there is only an insufficiency of the valve but no stenosis.

There are three general causes of disease of the aortic valve; namely, rheumatism, syphilis and arteriosclerosis. The young are apt to be rheumatic, the middle-aged, syphilitic and the aged, hypertensive or sclerotic. This division is quite arbitrary as many cases overlap from one age period into another. The frequency with which one type or another will be met will depend in a great measure on the locality where the physician practices. In certain sections of the country almost all cases of aortic insufficiency will be luetic in origin. This discrepancy



has made some authors state that aortic insufficiency is invariably due to syphilis. In a locality like New England, more individual cases of rheumatic aortic insufficiency are seen than any other type, although frequently this is associated with other valvular disease. On rare occasions aortic insufficiency may be functional or transient. This may occur during the very asthenic stages of marked anemia of the pernicious type and in hypertension.

### AORTIC INSUFFICIENCY

The two types of lesions of the aortic valve to be considered are aortic insufficiency and aortic stenosis. Let us first take up aortic insufficiency. The heart in this condition will become hypertrophied in the course of time and in some cases this enlargement becomes extreme. The left ventricle is the chamber that bears the main brunt of the leak and therefore it becomes not only thickened but eventually considerably dilated. This enlargement extends downward more than outward so that the apex impulse may be felt in the sixth interspace outside the nipple line or even lower. The apex impulse is apt to be heaving and forceful denoting a thickened musculature of the left ventricle. It is well to become familiar with this type of impulse no matter under what circumstances it is felt, for it gives a fairly satisfactory indication that the wall of the left ventricle is thickened and sometimes enables one to decide that the heart is hypertrophied, even when percussion outlines are doubtful. One gains the impression that with systole the apex impulse lifts the palpating finger and keeps it elevated for an appreciable time before it recedes. It does not merely tap the finger. Only on rare occasions can a diastolic thrill be felt at the base of the heart in aortic insufficiency. Percussion merely aids in estimating whether the heart outline is enlarged or not, and in no way helps in identifying aortic valvular disease. Auscultation reveals the decisive evidence of the diagnosis. In uncomplicated conditions, the heart rhythm will generally be found regular. Almost invariably a systolic murmur will be heard at the base of the heart and often there is a systolic murmur at the apex as well. The apical systolic murmur is either due to a relative mitral insufficiency or to a concomitant mitral endocarditis. When the aortic insufficiency is due to syphilis or sclerosis the apical systolic murmur should be regarded as due to relative mitral insufficiency and not to a true endocarditis because a true organic mitral insufficiency practically never results from syphilis or arteriosclerosis. Here the left ventricle becomes markedly dilated and the mitral ring, although essentially normal, is large and the leaflets do not completely close the opening during systole. When it is rheumatic in origin, it will always be difficult to decide whether or not the mitral valve is structurally diseased unless additional evidence is obtained pointing to mitral stenosis as well. If aortic stenosis is also present an apical systolic murmur may actually be coming from the aortic valve.



There are three possible explanations of the basal systolic murmur in aortic insufficiency. First, it may be due to an actual structural or relative stenosis of the aortic valve. This I think is more common than is recognized. Secondly, it has been thought that a roughening of the aortic wall such as is found in syphilis and arteriosclerosis may produce a systolic murmur. This I think is fallacious, for I have frequently heard no murmur when later at autopsy the aorta showed extreme degeneration. Finally, it may be due to the hyperdynamics of the circulation at that moment. There may be a momentary increase in the rate of flow of the blood during systole that accompanies aortic insufficiency. I believe that the latter factor is more important than is generally assumed.

The apical and basal systolic murmurs discussed in the foregoing paragraphs, although generally present in aortic insufficiency, are in no way diagnostic, for they occur when the aortic valves are competent. The most important and characteristic finding is the presence of a diastolic murmur heard at the aortic area, and even with louder intensity at the third left interspace near the sternum and propagated downward. This murmur has a blowing quality, starts directly with or replaces the second heart sound and is diminuendo in character. At times it is quite faint and requires most careful auscultation for its detection. In some cases it will be audible only if searched for with the patient sitting up and examined during deep expiration. In general it is unwise to make the diagnosis of aortic insufficiency without this finding.

Although the detection of an aortic diastolic murmur is valid evidence of aortic incompetency, especially if there are peripheral signs as well, postmortem examination may occasionally fail to confirm the finding of valvular disease. This does not mean that the diagnosis was incorrect, for the valve may have been incompetent in life and still show no structural disease at autopsy. When the heart is examined postmortem we do not see the structures as they existed with their normal tone and under blood pressure relations that prevailed during life. The valves may have been relatively insufficient because of dilatation of the aortic ring. Such relative aortic insufficiency is not uncommon in hypertension and in marked anemia when the valves are structurally sound. Similar dynamic dilatation has been repeatedly observed in the aorta itself. I have seen instances in which the *x*-ray showed very marked dilatation of the aorta, pointing strongly to the diagnosis of aneurysm, when at autopsy a perfectly normal small elastic aorta was found.

There are instances in which a presystolic murmur will be heard at the apex in cases of aortic insufficiency. This is the so-called "Austin Flint" murmur. It may be difficult to interpret the significance of an Austin Flint murmur, for it has the same time and quality and is heard in the same place as the murmur of mitral stenosis. It must be admitted, however, that this murmur can be present when the mitral



valve is not stenosed. The practical point of view that I have taken in this regard is that if the so-called Austin Flint murmur is heard in syphilitic aortic insufficiency, I would accept it as real, but that when it is present in a rheumatic case, I would be apt to regard it as due to concomitant mitral stenosis. This is based on the empirical pathological experience that syphilis never produces mitral stenosis and that rheumatism frequently does, even when aortic disease is also present. However, I have seen instances of rheumatic aortic insufficiency in which not only a presystolic murmur was present at the apex but also a presystolic thrill and x-ray evidence of a prominent left auricle were noted in cases in which at postmortem examination mitral stenosis was not demonstrated.

**Peripheral Signs.**—There are, in addition, very important peripheral signs of aortic insufficiency which are more or less dependent upon the increased pulse pressure that accompanies this condition. The most striking one of these is the hyperactive agitated pulsations of the arteries, especially the carotid arteries. They beat violently so that the entire lateral portions of the neck seem to pulsate. The radial pulse has a water-hammer quality, the so-called "Corrigan" pulse. There is a capillary pulse that may be made out on observing the finger nails while exerting mild pressure. This may also be detected in watching a flush come and go after rubbing the forehead or while pressing on the inside of the lower lip or the lower lobe of the ear with a glass slide. In addition, there is the so-called "pistol shot" in the femoral artery, that one may detect with the stethoscope. Here, if pressure is applied during auscultation, a systolic murmur may be heard. This murmur is really a normal phenomenon, but what is more significant is that in many cases of aortic insufficiency with appropriate pressure over the femoral artery, one may hear a distinct diastolic murmur (Duroziez's sign). These various peripheral evidences of aortic insufficiency are by no means pathognomonic. They occur in a less well-marked degree in a variety of conditions. Many states, in which there is a lax atonic peripheral vascular tree, may present these signs. They are commonly seen in association with hyperthyroidism, certain types of anemia, especially pernicious anemia, fevers, nervous hearts, and in some cases of hypertension. In most of these conditions the pulse pressure will also be found greater than normal. I have frequently observed the capillary pulse and a Corrigan pulse in conjunction with the above conditions, sometimes even noting pulsation of the retinal arteries.

The peripheral signs of aortic insufficiency, although in general not as helpful in diagnosis as the diastolic murmur, nevertheless are valuable when there are combined valvular lesions. This is especially true in rheumatic aortic insufficiency. Here, it may be difficult to interpret the origin of a diastolic murmur heard along the left sternal border. Such a murmur may have various causes. At times the diastolic murmur



of mitral stenosis may be so prominent and loud that it is actually well heard even toward the base of the heart. The diastolic murmur, on the other hand, may be due to an aortic insufficiency, which is combined with the mitral stenosis. It may be a so-called "Graham Steell" murmur. This latter murmur is supposed to be due to a relative pulmonary insufficiency occurring in mitral stenosis, and is heard in the pulmonary area (second left interspace) and along the left sternal border. Moreover, the presence of a diastolic murmur in this area together with the systolic murmur that is also present may be attributed to congenital heart disease. Finally it can be the result of organic tricuspid stenosis. Frequently it is impossible to make an accurate differentiation. In such instances the peripheral signs may decide the question as to whether aortic insufficiency is present or not. One may have to utilize all available means of diagnosis to arrive at a proper decision. The presence of auricular fibrillation will point definitely to mitral stenosis and the snapping quality of the first heart sound should make one suspect its presence. The *x*-ray finding of a dilated pulmonary artery will point to the diagnosis of a patent ductus arteriosus. When the patient has obvious aortic insufficiency and the question of an additional mitral stenosis comes up, if auricular fibrillation exists, both valves are probably involved. Also, if there is right ventricular preponderance in the electrocardiograms or a prominent left auricle on *x*-ray examination, it is likely that the lesion is not confined to the aortic valve, but that mitral stenosis is also present. For if aortic insufficiency were the sole lesion, we would expect marked left ventricular preponderance in the electrocardiograms. Although this evidence is not indisputable, it is helpful. In order to make the diagnosis of a Graham Steell murmur, it would be necessary to find this diastolic murmur present in the absence of peripheral signs of aortic insufficiency. One would also expect that the patient would be decompensated and that the murmur should either disappear entirely or at least diminish markedly in intensity as improvement in the circulation occurred. Although in most cases the interpretation of the basal diastolic murmur is not difficult, when it is doubtful it is apt to be of academic interest, for it may be assumed that except in extremely rare occasions a diastolic murmur means heart disease.

### AORTIC STENOSIS

Stenosis of the aortic valve is frequently due to rheumatic infection, is occasionally the result of arteriosclerosis, but is never syphilitic in origin. There was a time when the diagnosis of aortic stenosis was made much too frequently, as many patients who merely had a basal systolic murmur were regarded as suffering from this condition. This was followed by a period of undue caution when it was regarded as a very rare disease. At present we must take a middle course for there is ample experience to teach us that it is by no means rare and that it is frequently overlooked.



The younger individuals with aortic stenosis are generally regarded as suffering from rheumatic valvular disease, whether the aortic lesion is found to be an isolated one or is combined with mitral or tricuspid involvement. Individuals with aortic stenosis who are over fifty or sixty years of age, who frequently show no significant involvement of the other valves, have been regarded as arteriosclerotic. It has been my impression that many, if not most, of such patients are also rheumatic. It is curious that even in this older group a positive history of previous rheumatic infection will be elicited in almost 30 to 40 per cent of the cases. This is true notwithstanding the fact that the interval between the possible early infection and the present illness might have been forty to fifty years, that a history of rheumatism may easily have been forgotten, and that many years ago atypical rheumatic infections were doubtless overlooked. Furthermore, as a result of a better understanding of the pathological findings in rheumatism, pathologists are now willing to admit that cases which they formerly had called arteriosclerotic they are now ready to call rheumatic. Another argument against the arteriosclerotic origin of many of these cases is that as a rule the ascending aorta, where sclerotic processes in general are so extensive, is apt to show comparatively few atheromatous changes in aortic stenosis. This may, however, be due to a protective effect that the stenosed valve exercises in buffering the systolic impact of the aorta. It is also conceivable that some cases of aortic stenosis are the result of a previous non-rheumatic infection of the aortic valve, or may even represent the healed stage of a previous subacute bacterial endocarditis. Finally, the likelihood that arteriosclerosis alone can be responsible for a portion of the older cases cannot be entirely dismissed.

The general clinical features of aortic stenosis resemble those of aortic insufficiency in that it is more common in males, it is found in hearts with a regular rather than an absolutely irregular rhythm and is rarely associated with emboli or pulmonary infarction. The left ventricle is often enormously hypertrophied, with only very little dilatation. The heaviest hearts are those with aortic stenosis, although the largest silhouettes on x-ray examination are those of mitral stenosis. This is so because of the marked dilatation that occurs with the latter condition. Dyspnea comes late, the patients maintaining rather good health for long periods of time until congestive failure finally intervenes. When this occurs, the prognosis is grave, for death is apt to intervene within two years. The foregoing generalizations do not hold, however, when there is combined mitral and aortic valvular disease.

One frequent complication of aortic stenosis is angina pectoris. This, I believe, is often overlooked. It has currently been taught that angina is a common accompaniment of aortic insufficiency and little mention is made of its association with other valvular lesions. The explanation given is that the heart is nourished in diastole, for during the actual systolic contraction blood flows out through the aorta while



the coronary arteries are practically occluded. In aortic insufficiency blood is regurgitating back to the left ventricle from the aorta during diastole and with the diminished diastolic pressure there is inadequate nourishment of the heart muscle. Although this seems to be a logical explanation, there are certain conflicting clinical facts. In patients with aortic insufficiency the occurrence or severity of angina by no means follows the level of the diastolic pressure. Furthermore, in cases of syphilitic aortic insufficiency in which the mouths of the coronary arteries are not occluded the degree of regurgitation is very often of an extreme degree and yet angina is rare. On the other hand, it has seemed to me that in patients with aortic valvular disease and angina pectoris the element of stenosis has been more prominent than that of regurgitation. In fact, I have frequently seen angina with the former when there was no evidence of the latter.

The explanation of the frequency of angina in aortic stenosis is obscure. Possibly several factors are involved. It is now believed that there is coronary flow during systole as well as diastole. The ventricular wall is generally thicker in aortic stenosis than in pure aortic insufficiency or in other types of heart disease. This may result in a greater degree of relative myocardial anoxemia because the coronary blood supply may not keep pace with the hypertrophy, and oxygen diffusion into thick muscle fibers is more difficult. Furthermore, the work of the heart must be enormously increased to expel blood through such narrow valves. Also the velocity of the blood ejected through the stenosed valve must be terrific to maintain a normal output per minute. This rapid stream may possibly exercise a suction action on the mouths of the coronary arteries which lie just beyond the valve. If this happens, blood actually is extracted from, rather than fed to, the heart during systole. Finally, numerous cases have additional significant coronary sclerosis to account for the angina. However, I have seen young patients with aortic stenosis and angina in whom sudden death occurred, and postmortem examination showed normal coronary arteries. Here some of the above mechanisms may have been involved.

Angina is frequently overlooked in aortic stenosis because insufficient attention is paid to minor complaints such as mild sternal distress on hurrying. Often the patient will not even mention it to his physician and it will have to be brought out by direct questioning. This accounts for the frequent occurrence of sudden and unexpected death that is so common in aortic valvular disease. Granted that an integral part of the symptom complex called "angina pectoris" is a likelihood of sudden death, eliciting a proper history will often forewarn the physician of possible sudden fatalities in aortic cases that otherwise would remain unexplained. Furthermore, when no such previous history of angina can be obtained it is not inconceivable that a first attack of angina may end fatally. Not so long ago I saw a man of fifty-five who was suffering from congestive heart failure. After his condition had improved it was



planned that a complete thyroidectomy would be performed. I and many other physicians had seen him over a period of a year and we all were in accord in the diagnosis of aortic stenosis. Finally, on direct questioning, I found that he had a definite history of anginal distress of about two years' duration, a fact that he never mentioned before because his primary complaint was breathlessness. The day before the time set for the operation, while he was apparently in good condition, he suddenly expired. If the diagnosis of angina had not been made this patient's demise would have been regarded as one of those unexplained sudden deaths in aortic disease. Experiences similar to this one are not uncommon.

Another peculiarity of aortic stenosis is that it is frequently accompanied by a tendency to faintness or to actual syncope. The exact explanation of this is obscure but it may be linked up with peculiarities in the carotid sinus reflex. It is now well known that if this reflex is hyperactive, giddiness, weakness and fainting attacks can occur. It is, therefore, necessary to study the condition of this reflex in cases of aortic stenosis in the hope of throwing further light on this otherwise obscure phenomenon. So far, however, in most cases a normal sensitivity of the carotid sinus has been found. Syncope occurs particularly on effort and in the last analysis is probably due to cerebral anoxemia. It is unlikely that it is due to temporary heart block, despite the fact that conduction defects are common in aortic stenosis. May it be due to a temporary increase in heart rate with a decrease in cardiac output? The problem is still unsolved.

In this connection it is of interest that in aortic stenosis the heart rate is often slow. This may be so even when gross evidence of congestive heart failure is present. In fact there are few conditions apart from aortic stenosis in which advanced congestive heart failure will be found with a heart rate under 70 or 60, in which the slow rate cannot be accounted for by heart block or the administration of digitalis. This point has been useful in first directing suspicion to the diagnosis of aortic stenosis and then detecting the direct clinical evidence of the lesion by careful examination. This slow heart rate may possibly be due to the same factor that makes the patient subject to syncope or may in some way be related to the vagus apparatus.

The clinical diagnosis of aortic stenosis will in most cases depend on finding a systolic thrill at the base of the heart. When it is marked it can readily be felt in the second right interspace or over the upper or middle portion of the sternum. It is often missed because careful palpation is not practiced or because it is not searched for properly. A fainter thrill may only become apparent if palpation is performed during a held expiration with the patient sitting upright or leaning forward. It can be confused with systolic thrills that accompany congenital heart disease but the differential diagnosis is generally established without difficulty by the presence or absence of other findings



such as cyanosis, clubbing of the fingers, electrocardiographic changes (right ventricular preponderance with pulmonary stenosis and left preponderance with aortic stenosis) and *x*-ray examination. One must be careful to interpret as a thrill only that condition in which a real purr of significant duration is felt. In this way it will not be mistaken for systolic or other impacts of the chest that are common in hyperactive hearts or in thin-chested individuals. This differentiation is important, for true basal systolic thrills in the absence of congenital heart disease generally mean aortic stenosis.

A second indisputable sign of aortic stenosis is the finding of calcification of the aortic valve on *x*-ray examination. This is best detected by fluoroscopic examination but may even appear on the flat heart plate. It needs to be distinguished from calcification in the mitral valve or in the myocardium. This can be easily done by a trained observer, the main differential points being its location and the fact that at the very beginning of systole the calcification in the mitral valve will be seen to move upward whereas the aortic valve moves downward. It is obvious that calcification of a valve must be a late process, and when it is sufficiently advanced to become visible the valve must have been diseased for years. In fact, in many cases in which calcification is not very extensive it will not be detected. It is surprising, however, how frequently this diagnosis was made on *x*-ray examination and in all such cases that were examined postmortem the *x*-ray diagnosis was confirmed. Although calcified aortic stenosis is generally seen in older people it is not at all rare in younger persons. In the younger group the disease is obviously rheumatic in origin and I suspect that in many of the older group it is also of rheumatic source, although the older patients have been generally regarded as arteriosclerotic. It must be borne in mind that calcium deposits are often found in any long-standing chronic inflammatory process and if a patient survives an aortic lesion long enough calcification may result.

The other clinical signs of aortic stenosis are less important because they are more difficult to interpret. The second aortic sound is diminished in intensity or absent in many cases. The difficulty is that the pulmonary second sound may be audible in the aortic area making it impossible to distinguish the origin of these sounds. A systolic murmur will be present in practically every case of aortic stenosis. This murmur is best heard in the second right intercostal space (the aortic area) or not infrequently over the upper precordium and midsternal region. It is generally a fairly loud murmur and coarse in quality. The difficulty is that basal systolic murmurs are common in such a variety of circumstances that this alone cannot constitute reliable evidence for the diagnosis of aortic stenosis. When it is appreciated, however, that the constricting process, which eventually becomes sufficient to produce a palpable systolic thrill or calcification of the valve detectable by *x*-ray, is a very slow one extending over many years, it is evident



that the diagnosis can only be suspected in many instances during these intervening years. It is obvious that a very loud murmur must have been fainter or even very slight years before or at its onset. I am convinced that many patients who merely show systolic murmurs of moderate or loud intensity without much other evidence of heart disease have aortic stenosis, even when the systolic murmur is louder at the apex than at the base of the heart. I have followed such cases long enough to see the true nature of the lesion develop. At first a systolic murmur would be heard when there were no symptoms. Such a patient may have been refused insurance because of it. At this time a thrill, a diastolic murmur or appreciable hypertrophy would be absent. The murmurs might have been called functional systolic murmurs. I might have made the diagnosis of mitral insufficiency, because in some of these cases the murmur was quite prominent near the nipple as well as toward the base of the heart; or the murmurs may have been designated as instances of "systolic murmurs, cause unknown." Then as years went on a definite basal thrill would become palpable or calcification of the valve would be found on *x-ray* examination. Dyspnea or anginal pain were frequent eventual developments in such patients. The lesson to be drawn from these experiences is that we should be more ready to make the diagnosis of aortic stenosis before all the classical signs are present.

Insufficiency so frequently accompanies stenosis of the aortic valve that one should make a careful search for a basal diastolic murmur. When it is found it will lend support to the diagnosis that the aortic valve is at least diseased and that the systolic murmur, which can be due to so many other causes, is in fact the result of aortic valvular disease. Even the presence of a systolic and diastolic murmur at the aortic area does not necessarily indicate stenosis of the valve. Both murmurs are present in most instances of free aortic regurgitation without stenosis, such as occurs in syphilitic aortic insufficiency, a condition which never results in stenosis of the valve. It is sometimes taught that when there is stenosis of a valve there must be insufficiency, *i.e.*, if a valve is so deformed and scarred that it cannot open it would similarly be impossible to close completely. Although this view sounds logical and one is further impressed by its validity on seeing the rigid calcified valves postmortem, the fact remains that in many instances of aortic stenosis no diastolic murmur will be audible nor will there be any peripheral signs of aortic insufficiency. The same holds true for mitral stenosis, for here in many instances, although there may be a long diastolic rumble indicating a fair degree of stenosis, no systolic murmurs will be heard after the snapping first heart sound.

There are other auscultatory signs in aortic stenosis. Murmurs may be present at the apex of the heart indicating an additional organic involvement of the mitral valve or a relative insufficiency of that valve. The basal systolic murmur and thrill may be transmitted upward into



the vessels of the neck. The finding of a systolic murmur and thrill over the vessels of the neck, without similar signs over the chest, cannot be relied upon as certain evidence of aortic stenosis, because they may be present in other conditions such as hyperthyroidism, anemia and nervous states. Furthermore the apical systolic murmur may merely be transmitted from the aortic valve and may not signify any additional lesion.

The plateau form of radial pulse is fairly characteristic of aortic stenosis. It is often overlooked because the examiner does not think about it. When it is present the pulse will be found to rise and remain sustained longer than in other conditions. It will obviously be absent if there is also sufficient aortic insufficiency, for this will counteract the plateau character with a collapsing quality.

The blood pressure in aortic stenosis is variable. In the classical case the systolic level is apt to be low and the diastolic comparatively high producing a small pulse pressure. One frequently finds readings such as 110 mm. systolic and 90 mm. diastolic. Two other factors, however, affect the blood pressure so that almost any reading might be found in different cases, *i.e.*, an accompanying aortic insufficiency and an independent essential hypertension. The result is that both the systolic and the diastolic pressure may vary from very low to very high levels.

The *x*-ray in typical aortic stenosis will show a prominent, rounded left ventricle producing a boot-shaped heart. The electrocardiogram will reflect the degree of left ventricular preponderant hypertrophy that is present and occasionally disturbances in conduction.

### DISEASE OF THE TRICUSPID VALVE

Although functional insufficiency of the tricuspid valve is common, organic disease of this valve is rare. It is easy to understand the frequency with which this valve becomes relatively incompetent. When there is congestive failure, the heart is generally dilated. If the right ventricle dilates, the tricuspid ring is thereby stretched and the valve, although normal in structure, will no longer be able to close the enlarged opening. This will necessarily result in a regurgitation of blood from the right ventricle into the right auricle. This condition is common in all types of heart disease, but particularly in mitral stenosis, where the burden on the right side of the heart eventually becomes great. Under such circumstances, the patient is apt to have not only marked evidence of cardiac failure with cyanosis, edema, and engorged liver, but regurgitation of blood through the tricuspid valve may show itself in other ways. There may be an actual systolic pulsation of the veins of the neck and limbs, and a pulsating liver. It is no simple matter to detect these changes, for normally there are pulsations in the jugular veins which are difficult to time. Likewise it is difficult to distinguish systolic pulsations of the liver from pulsations of the right side of the heart and abdominal aorta. By placing both hands around the lower



right axilla and right upper quadrant of the abdomen, one may at times be able to sense an actual systolic expansion in the liver region with each heart beat as one hand moves forward while the other moves backward. In many cases the significance of a systolic pulsation is misinterpreted because it can be due to an impact from the neighboring abdominal aorta or the overlying heart. The most satisfactory clinical evidence of tricuspid regurgitation is the detection of faint pulsations in the veins of the forearms or forehead. As a result of tricuspid insufficiency, there is also a systolic murmur heard in the tricuspid area over the lower sternum. It is almost impossible to identify this murmur or to distinguish it from the systolic murmur of mitral origin heard at the apex that is invariably present and has the same quality. It probably is true that many patients with advanced cardiac failure have a functional tricuspid insufficiency even when no such diagnosis is made, for frequently at postmortem examination the tricuspid ring is dilated.

True tricuspid endocarditis is practically never a pure lesion. It has the same etiological background as disease of the mitral valve and in my experience it has invariably been associated with mitral stenosis with or without aortic stenosis. The diagnosis of organic tricuspid insufficiency or tricuspid stenosis is extremely difficult. Most authorities have considered it impossible to make this diagnosis. However, with the recent increased interest in this condition, more and more cases are being recognized. There are certain features which, although not pathognomonic, should arouse suspicion of the presence of tricuspid stenosis.

Patients with tricuspid stenosis have in general the same symptoms as those with mitral stenosis. Cyanosis in this condition is apt to be more intense, infarctions of the lung are very common and the liver is invariably enlarged. A striking feature in some of the cases is the fact that the liver may remain enlarged for a long time, even years, while the patient is comparatively comfortable and ambulatory. Ascites may also be very prominent and require frequent abdominal tapplings. The same thing may be true of hydrothorax, especially on the right side. Patients with tricuspid stenosis seem to tolerate these evidences of advanced heart failure with less dyspnea and general distress than do patients with other cardiac conditions. It is thought by some observers that in the course of time these patients are apt to develop a peculiar appearance of the skin, consisting of a slight olive discoloration. I have seen this in several instances and it has seemed to be a mild chronic jaundice with a slight faint greenish tint to it. It probably is the result of prolonged enlargement of the liver and no doubt may develop in other cardiacs when the same long-continued stasis takes place. Polycythemia is also a common finding in tricuspid stenosis. Abdominal symptoms are common, particularly pain and tenderness in the liver. What is more impressive, however, is the degree of comfort and the comparative absence of dyspnea and peripheral edema in the presence of a persistently enlarged liver and recurrent ascites. To this extent



it resembles constrictive pericarditis which is distinguished from tricuspid disease by the absence of significant enlargement of the heart or murmurs.

On physical examination the cervical veins are almost always found to be distended and prominent. These characteristics may occur in all types of right heart failure. The distinguishing feature in the tricuspid cases is that the condition is not only very marked but it persists for long periods of time, even when the patient is in a fairly good state of compensation. The same will be true of the venous pressure. It will not only be elevated, but with improvement it will not return as closely to the normal level as in other cardiac diseases, with the exception of constrictive pericarditis. The blood pressure is practically never significantly elevated in tricuspid stenosis (over 150 mm. systolic). Hypoproteinemia is common, probably the result of prolonged hepatic insufficiency.

Enlargement of the right side of the heart is very striking. The right auricle dilates tremendously and may even extend beyond the right midclavicular line. There is almost invariably a right ventricular preponderance in the electrocardiograms. Auricular fibrillation, although often associated with tricuspid stenosis, is not as frequent with the same degree of heart failure as in cases of mitral stenosis alone. This means that a fair number of these patients will show gross heart failure with a regular rhythm. The murmurs are similar to those that occur in mitral stenosis, but they are well heard over the lower sternum or to the right of the midline. When it is appreciated that in a series of thirty-two cases of tricuspid stenosis every case also showed well-marked mitral stenosis, difficulties in the interpretation of murmurs become apparent. When the systolic murmur and particularly the diastolic murmur are better heard near the midline than at the apex of the heart, it would lead one to suspect that the tricuspid valve is diseased. The frequent association of tricuspid stenosis with mitral stenosis, aortic stenosis and even adhesive pericarditis makes accurate diagnosis extremely difficult.

It is of considerable interest that amongst patients with chronic rheumatic valvular disease, those with tricuspid stenosis die at the earliest age. The age at death in these cases (thirty-four years) is about twelve years less than in those with mitral stenosis alone (forty-six years) and nineteen years less than in those with aortic stenosis alone (fifty-three years). However, the duration of symptoms or evidence of congestive failure is much greater in patients with tricuspid disease than in those in whom the other valves are involved. A recent study has shown comparative figures to be seven and five-tenths years for tricuspid stenosis, four and six-tenths years for mitral stenosis, and three and one-tenth years for aortic stenosis. In other words, although death occurs at the youngest age in the tricuspid cases, the patients with this disorder live longest once failure develops. The explanation



of this is that they have a mechanical embarrassment of the heart comparable to constrictive pericarditis, only the constriction is inside the heart at the tricuspid valve, and not outside in the pericardium. This process is more static and does not necessarily imply heart muscle failure. We, therefore, are witnessing in such cases evidence of right-sided failure, such as venous distention, enlarged liver and ascites, which has essentially a mechanical cause. Such congestion would obviously be much less serious or progressive than if it resulted from pure heart muscle failure.

It follows that if a case of known mitral stenosis carries on fairly well for years with evidence of right heart failure and shows persistently engorged cervical veins and enlarged liver, especially if this is out of proportion to the degree of dyspnea, and there is no hypertension, the diagnosis of tricuspid stenosis must be considered.



## 5

### DISEASES OF THE PERICARDIUM

DISEASE of the pericardium is almost always secondary to some other primary condition, whether it be an infection or some other morbid process. The pericardial abnormality may impair health or threaten life in one of several methods. As part of an inflammatory process, pus may form and give rise to the hazards attendant to a closed empyema. Sterile fluid, an exudate, a transudate or a hemorrhage may be so abundant that it produces cardiac tamponade, *i.e.*, interference with the normal movements of the heart. Finally, as a result of a previous infection or some other pathologic condition, scars, adhesions or bands may form in the pericardium impeding the free contraction or expansion of the heart. This latter process can conveniently be divided into two clinical types: *chronic constrictive pericarditis* and *chronic non-constrictive pericarditis*, the latter embracing the condition generally called *chronic mediastinopericarditis*.

The various common types of acute pericarditis will first be discussed. Then pericardial effusion and, finally, chronic constrictive and non-constrictive pericarditis will be considered.

#### RHEUMATIC PERICARDITIS

Acute rheumatic pericarditis generally develops about two weeks after the onset of the rheumatic infection. Its occurrence, however, is variable. At times it ushers in the rheumatic infection, actually preceding



any other manifestation of rheumatism. In fact, it may be the only evidence of the rheumatic infection, there being no joint pains whatever. On the other hand, it can develop after the original attack of rheumatism has subsided, just as one is ready to allow the patient to get out of bed. It is more commonly associated with aortic disease than with mitral stenosis. This may be explained when we consider the anatomical relation between the pericardium and the valves of the heart. At the base of the heart there is only the thickness of the aortic wall between the visceral pericardium and the aortic valves, while the entire musculature of the ventricles intervenes between the visceral pericardium and the mitral valve. If the process of infection is one of extension no matter in which direction, it is easily conceived how pericarditis would be more often associated with disease of the aortic than of the mitral valve.

**Symptoms.**—The symptoms of acute rheumatic pericarditis consist essentially of an accentuation of those symptoms that already exist as a result of the underlying rheumatic process. The heart, already rapid, becomes further accelerated so that a rate of 130 or more is not at all unusual. The respiratory rate becomes unduly rapid. Although this need not be associated with actual dyspnea, in some instances the rate of respiration may be 50 and the patient may be able to lie flat in bed. In most cases, however, there is actually some respiratory distress. The marked increase in the heart and respiratory rates is out of proportion to the degree of fever which often is no more than  $101^{\circ}$  or  $102^{\circ}$  F. Very frequently there is a rather peculiar and characteristic cough. This is short, hacking, irritative, and unproductive. Often there is pain in the chest. It was formerly thought that this chest pain was due to the inflammation of the pericardium, but there is now considerable doubt about this explanation. There are frequent painless instances of acute rheumatic pericarditis. Certain studies on human beings indicate that the pericardium for the most part is insensitive to pain. It may, therefore, be that the pain in pericarditis is due to an associated pleuritis. Notwithstanding the different possible explanations, pain in the heart region is fairly common in pericarditis. Occasionally the early pain is in the abdomen rather than over the heart and with the fever and leukocytosis that accompany it, an acute surgical condition of the abdomen may be simulated.

**Diagnosis.**—The diagnosis may be suspected from the evidence described in the preceding paragraph, but will finally rest upon detecting the to and fro pericardial friction rub. This is generally a harsh, grating sound, best heard in the third or fourth left interspace. It can remain localized or spread over a larger area. On auscultation, the sounds seem to be close to the ear and the intensity may be augmented by firm pressure with the stethoscope or by having the patient bend forward. This to and fro friction on rare occasions is quite transient, lasting only some hours, but generally it persists for days. When it is



loud, it seems to envelop the heart sounds rather than to follow them. When it is typical, it has two portions to it, one with systole and one with diastole, but I have seen instances in which for about twenty-four hours there was only a systolic element and later the diastolic portion was heard. At times, the continuity of the sounds is interrupted so that it may give one the impression that there are three or four portions to the entire friction rub. Because of the frequent association with aortic insufficiency, which has a to and fro systolic and diastolic murmur, it is sometimes quite difficult to distinguish a to and fro pericardial friction rub from the signs of aortic insufficiency. In fact, both conditions may be present. In some cases, it is necessary to delay judgment. If the to and fro murmur disappears, it must obviously have been due to a pericarditis. If it persists indefinitely, it is due to aortic insufficiency. The detection and proper interpretation of a pericardial friction rub is most important because it may be the only distinctive evidence that there has been an acute pericarditis.

There is another frequent development in acute rheumatic pericarditis that is helpful in diagnosis, particularly if by chance the pericardial friction was either missed or indistinct. This is the so-called "Ewart's sign." It consists of dulness and bronchial breathing heard below the angle of the left scapula. These findings are supposed to be due to atelectasis and compression of the left lower lobe of the lung from fluid accumulating in the posterior pericardial sac. It is not certain whether this explanation is true, but it is clear that the above signs which resemble pure lobar pneumonia frequently occur in acute rheumatic pericarditis. The bronchial breathing in this condition is apt to be quite loud, and generally is unassociated with any rales. The resemblance to pneumonia accounts for the frequent history obtained from rheumatic patients that they had pneumonia in childhood. I have seen numerous such instances in which the physician thought the child was suffering from pneumonia. Had not the proper diagnosis been made, such patients would have presented themselves in later years with aortic insufficiency in whom no other infection than pneumonia could be blamed as the cause of the valve disease. The development of Ewart's sign generally follows by some days the appearance of the pericardial friction rub. It may, however, actually precede it. I recall an experience in which such a case was shown as an instance of pure lobar pneumonia in a ward given up solely to the care of pneumonia patients. Because the patient had some of the secondary features of rheumatism discussed in Chapter 2, namely, repeated nosebleeds and a family history of rheumatic heart disease, and because he already had some aches in his joints, it was predicted that a pericardial friction would appear and other features pointing to rheumatic infection would develop. This occurred during the following days. I cite this case to illustrate the importance of accurate diagnosis and the indirect means by which proper diagnoses are sometimes made.



The third characteristic of rheumatic pericarditis is the frequent development of disturbances in conduction. Heart block is very common in this condition. When actual blocking of beats occurs, it is easily detected by auscultation. The rhythm, which is rapid and regular, will suddenly be interrupted by pauses. These pauses will not be preceded by a premature beat, which distinguishes it from extrasystoles. A complete heart cycle every now and then actually drops out. When the disturbance in conduction is less marked, no actual blocking of beats occurs. The heart remains perfectly regular. It is almost impossible to detect this change without graphic methods, although the presence of a gallop rhythm or a decrease in intensity of the first heart sound arouses one's suspicion. If an electrocardiogram or a polygram is taken, the conduction time as indicated in these curves will be found above the upper limits of normal which is two-tenths of a second. These slight changes are earlier evidence of what becomes heart block when the process is more marked. Disturbances in conduction indicate a simultaneous myocarditis which frequently accompanies pericarditis. Except for occasional cases of coronary thrombosis, this is the only form of pericarditis in which conduction disturbances are apt to occur. Apart from the friction sound, the Ewart's sign and the conduction disturbances, there is little that is distinctive on physical examination in cases of rheumatic pericarditis.

It is likely that in most cases of rheumatic pericarditis an effusion of a greater or lesser extent develops. When it is slight, it is impossible to detect it. The inflammation gradually subsides and the fluid is absorbed. The pericardial friction disappears after lasting a few days, but the signs of compression of the left lower lobe of the lung and the conduction disturbance may persist for two weeks or more. Gradually both of these signs also disappear, although on rare occasions the electrocardiograms may continue to show a delayed conduction time indefinitely. Changes in the ventricular complexes of the electrocardiograms may also be present (Chapter 21, Fig. 121).

**Prognosis.**—After a prolonged illness, generally lasting months, the patient recovers. The prognosis in rheumatic pericarditis in general is good. The immediate mortality is about 15 per cent. The physician should always keep before him the possibility and even the likelihood of a favorable outcome in the face of what seems to be a very stormy disease. One must appreciate that we are dealing primarily with an infection which at this time is in an acute fulminating stage and that the acute process can subside. Patients may be desperately sick and make an excellent recovery even if signs of congestive heart failure develop. Acute rheumatic pericarditis practically never leads to constrictive pericarditis.

The ultimate outcome, if immediate recovery takes place, will depend in a great measure upon whether the valves have been affected during the acute process. If during the attack of pericarditis no diastolic



murmur develops, recovery may be complete from a symptomatic point of view. The presence of a systolic murmur will have the same significance as systolic murmurs in general (this matter is discussed in detail in other chapters). I have seen numerous instances in which, after a most violent and desperate attack of rheumatic pericarditis, recovery was complete and the patient was subsequently able to carry on even strenuous physical work. In such cases there may even be no evidence of heart disease whatever in later years. The subsequent health of the patient and ability to work are quite different when evidence of valve disease develops. Such experiences have impressed upon me most forcefully the importance of the valves in heart disease. Recovery from injury to the myocardium after acute infections seems to take place most satisfactorily, but when the valves are structurally involved, in many cases there appears to start a progressive vicious cycle, with subsequent development of circulatory insufficiency.

Great emphasis has rightly been placed on the importance of the myocardium in heart failure. This applies primarily to non-valvular heart disease. In chronic rheumatic heart disease a fairly healthy ventricular musculature is noted on postmortem examination. If the myocardium rather than the valves were of primary importance in rheumatism, it is surprising that we do not see patients in later life, who have had rheumatic fever in childhood, dying of heart muscle failure without valvular disease. Apart from the fatalities that occur during the acute rheumatic carditis when the state of the myocardium is all important, death due to heart damage in subsequent years is limited to those patients who develop valvular lesions, while those who recover from the acute infection without murmurs or without valvular injury may never have any further trouble from the heart. The degree of valve deformity is not the sole final cause of failure of the circulation, although it is a most important factor in the disability; the so-called "accidents" of heart disease intervene like bacterial endocarditis, infarctions, emboli and infections. When these accidents do not occur then the valvular damage present at the time of heart failure will be found to be adequate to explain the situation without recourse to any significant role played by the heart muscle.

**Treatment.**—The treatment for a patient with rheumatic pericarditis comprises little more than that employed for the underlying rheumatic fever. It is customary to give salicylates either by mouth or by rectum as has been described in a former chapter. Some believe that large doses of salicylates actually produce absorption of the pericardial effusion. Digitalis will have no beneficial effect unless there is evidence of congestive heart failure. The patient, of course, should be made as comfortable in bed as possible and requires careful nursing to spare him any unnecessary effort. He generally will find it more comfortable to be in the semirecumbent or at times in the upright position. Codeine



in doses of  $\frac{1}{4}$  to  $\frac{1}{2}$  grain may be given every four hours, even to children, for precordial pain or cough. The only additional therapeutic measure that may be helpful is the use of an icebag over the precordium. This sometimes alleviates the heart pain and diminishes the sensation of palpitation. In only a very rare case will it be necessary to tap the pericardium. This may be a life-saving measure and will be taken up under the general discussion of pericardial effusion.

### PERICARDITIS IN PNEUMONIA

There are certain distinctive features in the pericarditis that occurs with pneumonia. In the first place, it is not a common complication of pneumonia. Secondly, it occurs almost exclusively in those cases of pneumonia that have empyema of the pleura. Furthermore, it is more commonly associated with empyema in the left pleural cavity than in the right. In fact, if a diagnosis of pericarditis is made in a patient with pneumonia, empyema of the pleura, especially on the left side, should also be sought for. Pericarditis, when it does occur, is apt to develop in the latter days or after the crisis or lysis would be expected. A most important difference between the pericarditis in pneumonia and in rheumatic fever is that the pneumococcus is likely to produce pus and rheumatic fever never produces pus. For this reason the diagnosis of the former is important.

The diagnosis of pericarditis with pneumonia will rest almost entirely upon detecting the pericardial friction rub. When this occurs and is heard its interpretation is a good deal more simple than in rheumatic fever, for there are no endocardial murmurs to confuse the picture. If the pericardial friction rub is not heard, pericarditis may yet be suspected in occasional cases of pneumonia if during the second week of the disease there develops a left pleural empyema which is satisfactorily treated and yet a septic course continues. One must remember that a patient suffering from pneumonia may have an empyema cavity in the pleura, well cared for, and yet die of an empyema of the pericardium. If there is any suspicion of this, an exploratory puncture of the pericardium is indicated, because the effusion under such circumstances is purulent and not likely to heal spontaneously. If pus is found in the pericardium, surgical drainage should be instituted. This complication of pneumonia is a most serious one. The outlook is practically hopeless if the pericardium is not drained although recovery may take place under surgical treatment.

It is not unlikely that with the use of sulfa therapy instances of pneumococcus pericarditis may actually heal without surgical drainage. May not some of these cases make up the ground work upon which subsequent constrictive pericarditis develops? Pericarditis developing during virus pneumonia, however, although also rare, does not appear to produce pus and will not necessitate aspiration or drainage.



## TUBERCULOUS PERICARDITIS

Tuberculous pericarditis is very rare in general practice. Its mechanism and development are quite similar to tuberculous pleurisy. In both conditions the process may be dry or fibrinous, or a serous or serousanguineous effusion may be poured out and adhesions may develop. Tuberculous pericarditis, however, is a much more serious complication of tuberculosis. It is always secondary to tuberculosis of the pleurae, lungs or mediastinal glands. The diagnosis of this condition in the early stages will depend almost entirely on the detection of a to and fro pericardial friction rub as in other types of acute pericarditis. There will be no evidence of either heart muscle disease or of valvular disease, for the heart proper is very refractory to the tuberculous infection. The striking characteristic of tuberculous pericarditis is a tendency to large pericardial effusions and it is the one condition that may require repeated tapplings. The exudate will be found to be sterile on ordinary bacteriological examination, just as occurs in rheumatic effusions. Here, on the other hand, inoculating some of the fluid into a guinea-pig may prove that it is tuberculous or occasionally the tubercle bacilli may be found on staining part of the fluid. Whenever a large effusion is found (800 to 1200 c.c.), especially if there is a tendency for reaccumulation, tuberculosis should be suspected. Effusions are apt to be larger than in rheumatism because they are poured out slowly, allowing the heart and pericardial sac gradually to accommodate itself to the increased pressure, and because the heart itself is not diseased. In rheumatic pericarditis, the reverse is true. The accumulation of the fluid is more rapid and either the heart muscle or the valves are generally affected. The heart then would have failed before such extensive effusions as occur in tuberculosis could have been poured out.

It is quite likely that many cases of tuberculous pericarditis are unrecognized, run a mild course and heal, thereby forming the basis of chronic constrictive pericarditis in later years. In some instances evidence of constrictive pericarditis may develop within a few months after the first detection of acute pericarditis. There may not be a large pericardial effusion and what fluid there is may be pocketed and not free.

## PERICARDITIS WITH CHRONIC NEPHRITIS

Pericarditis is a very common complication of chronic nephritis. Its exact nature is not altogether clear. Although it probably is due to a terminal infection, in some cases bacteriological examination of the pericardium and pericardial fluid at autopsy shows no evidence of any infectious organisms. In other cases, streptococci are found. It comes insidiously in those who have advanced chronic nephritis with marked nitrogen retention. There frequently is a slight fever and leukocytosis with the pericarditis. These need not be present. The disease may be entirely painless. The diagnosis will rest on the finding of the pericardial friction rub. It is only important from the point of



view of prognosis for it denotes quite decidedly that the end is near. Out of twenty-four cases of pericarditis and nephritis that I once studied, in twenty-three instances death occurred within three weeks. The other patient was practically moribund when he was last seen. Pericarditis sometimes develops in an ambulatory patient who has advanced chronic nephritis and even here it indicates that a fatality is to be expected within one month. There is no therapy whatever that can be offered for this condition, for it is the underlying nephritis with its uremic manifestation that is the real problem. While following a patient with advanced chronic nephritis, it is important to watch for acute pericarditis purely as an aid in estimating the prognosis, for sometimes such information is needed in giving certain advice to the family and patient.

### PERICARDITIS WITH CORONARY THROMBOSIS

A more detailed discussion of coronary thrombosis will be taken up later (Chapter 6). At this point the single feature of pericarditis which frequently develops in coronary thrombosis needs to be considered. Following an attack of partial or complete occlusion of a coronary artery, the part of the ventricles supplied by this vessel becomes infarcted. When the process of infarction is sufficient to extend from within the ventricle to its surface, the pericardium is necessarily involved. At this point, a localized fibrinous exudate develops and therefore a true serofibrinous pericarditis will result. It is obvious that if the infarction does not extend to the visceral pericardium, no pericarditis occurs; or if the site of the lesion is in the posterior part of the heart or over the dome of the diaphragm even if the pericardium is involved, a friction rub might not be audible. Furthermore, the friction rub that does develop with coronary thrombosis can be very faint and transient, and, therefore, can easily go undetected. This finding will be more common the more frequently and the more carefully patients with coronary thrombosis are examined but will not be detected in more than 10 to 20 per cent of the cases.

Generally in one to several days after the onset of the attack, the friction rub becomes audible. Sometimes it appears later when the patient is already free from the agonizing pain that was present in the early days. It does not add any additional symptoms to what the patient already has and it does not appreciably alter the prognosis of the condition or the treatment, except in so far as it indicates a significant area of myocardial infarction. It is a helpful sign in making the diagnosis in some doubtful cases. The fact that there is a fever and a leukocytosis does not mean that an infection of the pericardium is taking place, for these result from the original infarction of the heart muscle and may be regarded as a reaction to the absorption of foreign protein. Only on very rare occasions is pericardial effusion associated with this type of pericarditis.



## MISCELLANEOUS FORMS OF PERICARDITIS

We have just discussed the five most common conditions in which inflammation of the pericardium takes place. There remains a heterogeneous group of conditions in which the pericardium may become the site of inflammation or of the accumulation of fluid. Any generalized sepsis may localize in the pericardium. It is then likely to be a terminal event. There is one type, not extremely rare, in which pericarditis due to streptococcus infection occurs with the development of a purulent effusion. This really is a part of a more general streptococcus infection and is especially associated with an antecedent sore throat. There are also instances of acute pericarditis that may be called idiopathic, for they do not fall into any of the forementioned groups. They present the picture of an acute infection, possibly virus in origin, do not produce empyema, show no stigmata of rheumatism, but may resemble acute coronary thrombosis, because of the chest pain and the presence of minor electrocardiographic changes. This condition runs a favorable course and recovery is complete. Furthermore, in any cachectic state such as advanced carcinoma, leukemia or pernicious anemia, when the red blood cell count is extremely low, fluid may accumulate in the pericardial sac as it does in other cavities of the body. This was quite common, in the days before liver was used, during the terminal stage of pernicious anemia. A non-bacterial type of pericarditis has recently been recognized as a frequent complication of lupus erythematosus disseminatus, a condition which may simulate rheumatic fever very closely. Finally, fluid not infrequently collects in the pericardial sac in cases of generalized anasarca from circulatory failure. Here it is a part of the process of edema which produces ascites, hydrothorax, and hydropericardium. When fluid accumulates in the pericardium with heart failure or in advanced anemic states, it is really not due to an inflammation of the pericardium, but is rather a serous transudate. Its diagnosis and treatment will be taken up under the discussion of pericardial effusion.

## PERICARDIAL EFFUSION

Fluid may accumulate in the pericardial sac in the form of an exudate resulting from inflammation or as a transudate resulting from alteration in the circulation of the blood. The latter type can properly be called hydropericardium. The diagnostic criteria for the presence of fluid in the pericardium are essentially the same no matter what type of fluid is involved. Accumulation of less than 200 or 300 c.c. in an adult produces such minor changes that it cannot be detected clinically. The only condition in which it would be important to recognize such a small amount of fluid is when there is empyema of the pericardium such as may occur in pneumonia. Here if there is some reason to suspect its presence, the fluid should be sought for by an exploratory puncture.

As the amount of fluid increases, certain physical signs may develop.



The area of cardiac dulness enlarges. The change in the outline of the heart from day to day is important, for the actual increase in the dimensions is more significant than the fact that at any one time there is enlargement of the cardiac area. A physician who observes the patient from day to day should, therefore, keep accurate data as to the position of the borders of the heart. Dulness on percussion will extend outwards to the left, although the apex impulse may remain in the same position and, in fact, move gradually inward. The presence of appreciable dulness to the left of the impulse is a valuable sign. More important still is increasing dulness in the left upper region of the heart. This normally is made out at the third interspace or third rib, but in pericardial effusion it may extend as high as the second interspace or even higher. Submanubrial dulness also increases, and the right border of the heart extends outward so that with large effusions it may reach the right nipple line. Some years ago, it was thought that an obtuse cardiohepatic angle was a very valuable sign of pericardial effusion. It has been proved by experiments on cadavers and by careful *x*-ray examination of living patients that this is not generally true. I have withdrawn as much as 900 c.c. of fluid from the pericardial cavity of patients in whom an *x*-ray had shown that the cardiohepatic angle was still acute. The general contour of the heart as a result of the effusion becomes more globular and the base of the heart which is made up of the aorta and the great vessels becomes less elongated, and more rounded to conform with the general spherical configuration of the entire shadow. As has been mentioned, in rheumatic cases there may be evidence of atelectasis of the left lower lobe of the lung. The left lobe of the liver is thought by some to descend as a result of pericardial effusion. I have not found this sign of much practical value, for even if the liver is palpable it may be the result of hepatic engorgement and cardiac failure rather than as a specific downward displacement produced by the pericardial effusion.

On auscultation, the heart sounds may be found to be muffled and diminished in intensity. It is often thought that if fluid accumulates in the pericardium, the pericardial friction sound disappears. This is by no means necessarily true, for frequently large effusions will be seen with the persistence of a loud to and fro rub. It has been quite definitely shown that, in some cases of pericardial effusion, there is a true pulsus paradoxus. Here the pulse which is already small, may diminish markedly or actually disappear with inspiration. Pulsus paradoxus has been observed in cases of pericardial effusion and found to disappear with the removal of the fluid only to return when the fluid reaccumulated. An interesting observation in this regard was made some years ago showing that most normal individuals may make the pulse disappear in the radial artery by throwing the clavicle backward entirely independently of the respiratory cycle. By this manipulation, the subclavian artery is compressed, obliterating the pulse wave to the arm. With normal inspiration, the clavicle is thrown backward to some extent. This



mechanism is probably in a measure responsible for the production of pulsus paradoxus. The other clinical findings that a patient with pericardial effusion may present are those that are more intimately associated with the underlying disease such as rheumatism, pneumonia, leukemia and the like.

*x-Ray examination* serves as a most valuable aid in this diagnosis. In general, it confirms more accurately the heart outlines, and gives a better picture of the peculiar globular shape taken on by the heart shadow. A further point that is obtained in this way is the lack of demarcation between the left auricle and left ventricle which ordinarily is made out either fluoroscopically or by flat *x-ray* plates. On fluoroscopic examination, the individual contractions of the various chambers of the heart are difficult to distinguish, and instead, indistinct wavelike movements are seen at the outer portions of the heart. The configuration of the heart will also change materially if two plates are taken, one with the patient recumbent and one upright. The shadow at the base of the heart will be found broader in the former than in the latter. Although *x-ray* examination is extremely helpful, it is by no means infallible. The differential diagnosis generally consists in distinguishing a pericardial effusion from a grossly enlarged or dilated heart. This is occasionally quite difficult and at times the only proof will come by making an exploratory puncture of the sac.

**Pericardial Tapping.**—The question now comes up, when and where should we perform a pericardial tapping? The vast majority of cases with rheumatic pericardial effusions should not be tapped; they so frequently do well if left alone. If there were no risk in tapping the pericardium and it were as simple a procedure as a puncture of the pleura, it probably would be helpful in shortening the illness of many patients with rheumatic pericardial effusion. But there is danger of an immediate fatality. The danger does not consist merely of piercing the heart. In the experimental animal, heart's blood may be obtained repeatedly by puncture without any deleterious effects. Another danger lies in piercing one of the coronary vessels. These lie very superficially just below the visceral pericardium and have no supporting tissue covering them. If one of these vessels is punctured there may be a continuous gradual ooze with a fatal hemorrhage into the pericardial sac. I have seen two instances in which fatalities occurred. There are times, however, where tapping a rheumatic patient is indicated and may possibly be life saving. If the general condition grows worse, the stage may be reached when tapping seems safer than allowing the fluid to remain. Exactly how to estimate this point is difficult to describe, and will depend more upon judgment and general experience than upon any single indication. If the systolic blood pressure is still maintained satisfactorily it is generally safe to delay. It is surprising how extremely sick such a patient may be and recover satisfactorily without tapping.

There are various sites for performing a pericardial puncture. Dif-



ferent physicians prefer different procedures and I judge that it is a matter of custom rather than that one method has any advantage over another. The method I prefer is to go in at the fifth left interspace, just inside the border of dulness and outside the apex impulse. It is well to cocaine the skin at this point. The needle is inserted inward, backward and slightly upward. One should select a trocar with a dull point to avoid unnecessary scratching and piercing of blood vessels. It should be pushed in slowly and it is better to have it attached fairly intimately to a syringe rather than to a suction bottle, for it is then more easily manipulated, and by frequent trials with the plunger of the syringe, one can more quickly and instantaneously tell when fluid is obtained. If the needle is pushed in very gradually, applying frequent suction, fluid may be obtained even without ever feeling the pulsations of the heart. If these are felt with the tip of the needle, it should immediately be slightly withdrawn and suction continued. When fluid is obtained, it should be removed slowly and as much should be taken out as comes freely. It is not wise to spend time looking for fluid if none is obtained at the outset, because it is this aimless exploration which may prove disastrous. The danger in the puncture is greater if it turns out that there is no pericardial effusion than if a large amount of fluid is present.

Another method for pericardial puncture is the so-called "Marfan's procedure." This consists of inserting the needle just below the lower border of the ensiform cartilage, going upward, backward and inward. This avoids the peritoneal cavity and enters the pericardium from above the diaphragm. Another method is to reach the pericardial sac from the right of the sternum, inserting the needle in the fourth interspace about 1 inch from the right sternal border. Finally, some clinicians prefer to puncture the pericardium from behind and insert the exploratory trocar below the angle of the left scapula. On one occasion I removed 500 c.c. of fluid from the pericardium by this last method after no fluid had been obtained by exploring through the fifth left interspace. In general, it may be said that tapping the pericardium should be regarded as a major procedure and should not be undertaken except after the most careful consideration.

In cases of rheumatic effusion, it is practically never necessary to tap the pericardium more than once. On occasions, however, within a day or two after the tapping, extensive effusions will develop in the left pleural cavity. It is not unlikely that when this occurs, the fluid actually continues to be poured out from the pericardium through the hole made by the trocar into the pleural space. I have seen two instances where this occurred, and during this time the patient seemed temporarily a good deal worse than before the pericardium was tapped. In each instance, marked improvement resulted after tapping the pleural cavity and removing about 2 liters of fluid.

Tuberculous pericarditis with effusion frequently requires repeated



tappings in contradistinction to rheumatic pericarditis. Favorable results have been reported from the reinjection of air after the removal of the fluid. It has been recommended that when this is done, the volume of air injected should be about half the volume of fluid removed. In hydropericardium occurring in cachectic conditions or with advanced cardiac failure, there is little to be gained by tapping the pericardium. It is conceivable that under certain circumstances, the patient's life may be slightly prolonged by releasing the pressure on the heart produced by a tense pericardial sac, but unless there is hope that the underlying condition may be improved subsequently, the relief will be only very brief. The finding of bloody fluid in the pericardium, not produced by trauma, should make one think of a neoplasm of the pericardium.

### CHRONIC CONSTRICTIVE PERICARDITIS

The type of chronic pericarditis now to be discussed, *i.e.*, constrictive pericarditis, is of particular importance because it is amenable to surgical treatment. As a result of some previous infection or inflammatory reaction in the pericardium a slow progressive fibrosis results, often with considerable calcification, that produces a constricting influence on the movements of the heart. In the course of months or years the heart may become greatly embarrassed so that it is unable to expand or dilate properly. It is obvious that if the right side of the heart is prevented from performing an adequate diastole or dilatation, it cannot receive the normal amount of blood. It follows, therefore, that the heart cannot expel an adequate amount of blood in any given period of time, for it cannot propel more than it receives. The result is a decreased cardiac minute output. Because of the impediment to a free inflow into the right heart the venous pressure or back pressure increases. This affects the superior and inferior cava as they both empty into the right auricle. The pressure in the pulmonary vessels may not be affected unless the restriction in the motions of the left auricle and ventricle is greater than that of the right, which is not usually the case. The disturbances in the dynamics of the circulation just mentioned take place irrespective of whether the ventricles are capable of normal systolic contractions or not. Even if there were no other abnormalities, such as myocardial, valvular or hypertensive disease, and the heart were otherwise perfectly normal, pericardial constriction would necessarily produce these changes. There often is another disability resulting from these pericardial scars that makes it more difficult for the heart to contract after it has dilated. The pericardium may be bound down to neighboring firm structures, such as the ribs, and the work of the heart thereby increased. This handicap in contraction is much less important in cases of constrictive pericarditis than is the difficulty in expansion.

The etiological factor in this condition is not always discernible. A large number of cases are due to tuberculosis, some to a staphylococcus



and others to a pneumococcus infection. Other undetermined infections, originally minor in nature, may prove to be the cause of some of the cases that at present have not been classified. It is quite evident that in many cases no definite past history of acute pericarditis can be elicited. If the pneumococcus and streptococcus have been playing a frequent etiological role in the past, realizing that such types of acute pericarditis formerly were very fatal, it may be predicted that with current sulfa therapy, in many more acute cases recovery will take place and possibly form the background for a larger number of chronic cases of pericarditis in future years. A most impressive fact in considering etiology is that rheumatic fever, such a common cause of acute pericarditis, is practically never the cause of chronic constrictive pericarditis.

**Symptoms.**—Let us now consider the symptoms and clinical findings in constrictive pericarditis. Such patients may complain of weakness, shortness of breath, abdominal distress and, later, swelling of abdomen and legs. The amount of embarrassment that will be present and the severity of symptoms will obviously depend on the degree of constriction around the heart and the stage at which the patient is being observed. In general there are two types of conditions that it might imitate and with which it often is confused. Because of breathlessness, fatigue and cardiac findings, it may resemble the other more common forms of heart disease and because of the enlarged liver and possibly ascites it may erroneously be regarded as cirrhosis of the liver.

**Diagnosis.**—Physical examination of the heart often reveals nothing abnormal. The rhythm is generally regular, although in a small number of patients auricular fibrillation may be detected. Gallop rhythm is not uncommon. No murmurs are present or only a slight systolic murmur may be heard. Occasionally a faint, prolonged third heart sound is audible at the apex resembling a faint murmur of mitral stenosis. The absence of other evidence of mitral stenosis, such as a dilated left auricle and an accentuated first heart sound, should enable one to distinguish the two conditions. The heart is quiet and inactive. The customary apex impulse is absent or feeble. One of the most significant findings is the absence of any appreciable cardiac enlargement. In almost all other forms of congestive heart failure (with which this is confused) the heart is enlarged. This is best determined by *x*-ray examination, which is discussed below. The blood pressure is always within normal limits or even lower than normal and the pulse pressure tends to be decreased. The pulse is small, somewhat rapid, and frequently shows a pulsus paradoxus. This latter finding consists of a marked diminution or disappearance of the pulse with inspiration, and can be elicited by palpation of the radial artery or better still while ausculting during blood pressure determination.

The liver is always enlarged and generally palpable, and in later stages ascites may develop. In cases of long standing, repeated ab-



dominal taps may have been performed. For a considerable time peripheral edema is likely to be absent but eventually swelling of the legs will appear. Probably, as a result of the prolonged hepatic congestion, hypoproteinemia may be present and this in turn helps to produce a stubborn form of edema. Unlike other types of heart disease, constrictive pericarditis is characterized by breathlessness only late in the course of the disease and paroxysmal dyspnea or orthopnea does not occur. The pulmonary findings may, therefore, be inconspicuous, unless evidence of pulmonary tuberculosis is present. Eventually, however, true congestion of the lungs and even hydrothorax may appear. The electrocardiograms may be of low amplitude and often show flattening or inversion of the T waves in Leads I or II.

The venous pressure is increased. This is detected by noting the distention of the cervical veins while the patient is sitting upright or in the semirecumbent position, when normally the veins ought to be collapsed, or in finding distended veins on the dorsum of the hand or forehead. Venous pressure in the arms should be determined and will always be found elevated, generally over 200 mm. This increase in venous pressure will also be found in any case of right-sided heart failure, no matter what the cause may be, and especially in tricuspid insufficiency or stenosis. The striking point in constrictive pericarditis is that the venous pressure is elevated when the other evidences of heart failure are not very impressive; it remains elevated even after vigorous methods are employed that are ordinarily helpful in cases of heart disease. Finally the pressure both in the veins of the arms and legs is elevated, whereas in cases of cirrhosis of the liver with ascites or in some other abdominal conditions the increase in pressure will be confined to the veins of the legs.

An interesting alteration in the dynamics of the circulation is decrease in the minute output of blood. What is even more significant is that whereas a normal heart can increase its output per minute five or ten fold with physical effort here the mechanical handicap prevents any such response. This explains the fact that these patients may be comfortable at rest but become markedly fatigued on effort or actually find it impossible to do very much physically.

The *x*-ray findings are often of primary importance in the diagnosis of constrictive pericarditis. On fluoroscopic examination the heart beat is found to be quiet. The movements of the cardiac borders are diminished. This decrease in contraction of the cardiac chambers, although visible to some extent throughout the heart, is often more marked over certain areas. Careful search should be made particularly in the region of the right auricle and ventricle, for this is the favorite site for fibrotic plaques to produce constriction. Kymograms may furnish permanent records of the actual amplitude of contraction of the cardiac chambers and serve for comparison in judging postoperative results. Furthermore, in many instances, calcification of the pericardium



even to an amazing degree may be detected. In some cases, especially those due to tuberculosis, calcification of the pleura may also be found. It was previously stated that the heart is not enlarged. That is certainly the general rule, but occasionally because the pericardium is so thick, or because of pockets of fluid, the cardiac silhouette may be increased. Although the *x*-ray findings are very valuable in detecting disease of the pericardium, whether or not the condition causes constriction of the heart must depend on other observations, especially the increased venous pressure, for there are instances of marked pericardial calcification in which the dynamics of the heart are not disturbed.

Once the possibility of constrictive pericarditis has been considered, the foregoing features and examinations enable the physician to make the diagnosis. The reason that these cases are often not detected is that the condition is not thought of and will be confused with two general groups of other cases. It is amongst patients who are regarded as having heart failure, in whom the ordinary causes are lacking (*i.e.*, hypertension, coronary sclerosis, valvular disease, hyperthyroidism, etc.) or who are thought to have cirrhosis of the liver, in whom constrictive pericarditis will be found.

Another aid in diagnosis is the response to medical treatment. Whereas most cardiacs with congestive failure improve on rest, digitalis and diuretics, patients with constrictive pericarditis only occasionally show any appreciable response. The venous pressure may fall slightly but will remain distinctly above normal despite all efforts. In this respect the condition resembles tricuspid stenosis, from which it can be distinguished by the absence of any valvular disease or marked cardiac dilatation.

One may repeat that constrictive pericarditis needs to be sought for amongst patients who, generally twenty to forty-five years of age, appear to have some form of heart disease or cirrhosis of the liver, who have a small, slightly rapid, often paradoxical pulse, generally regular in rhythm, although occasionally grossly irregular, who have no hypertension, significant murmurs or cardiac enlargement, and who always show an increased venous pressure and an enlarged liver.

**Treatment.**—The treatment for patients with this condition is surgical. Some temporary improvement may result from medical management, mainly from diuretics, restriction of salt and fluid intake. Digitalis is rarely of value and may aggravate the condition. When the protein content of the blood is low a high protein diet should be given and infusions of plasma may be helpful. Inasmuch as the fundamental problem is mechanical, surgical resection of the pericardium (Delorme's operation) is the only satisfactory means of relief. The surgeon should be one who is specially expert in cardiac surgery for the operation is difficult and not without risk. Owing to the work of White and Churchill, Burwell and Blalock, Cutler and Beck, and others, sufficient progress has been made so that operative results are now very satisfactory.



Some patients are actually cured and many others are considerably relieved. A few are no better because the involvement is too extensive and the surgeon is unable to free enough of the heart to permit adequate expansion. Rarely the lung is similarly bound down to the thorax, so that respiratory failure continues despite a satisfactory restoration of cardiac mobility. Occasionally secondary operations are necessary because sufficient relief does not result following the first attempt. Finally, the physician should be patient in appraising the operative result. Although some subjects may make a dramatic recovery even in a few weeks, others show little gain for many weeks or even months and yet finally make satisfactory recovery.

In performing the operation due regard must be paid to the eventual dynamic relations of the circulation. If the right side of the heart is freed of constrictions and enabled to fill normally and the left ventricle remains bound down, the situation may be no better or even become worse than it was before. Now, added pulmonary congestion may develop because of the inability of the left ventricle to receive or expel its quota of blood. In other words, the dissection must be carried out so that a balanced state of the circulation results. In fact, it would seem wise to free the left side before the right so that even during the operation acute pulmonary edema might be prevented. One need have no fear of the ability of the heart to take care of the increase in work, since, in cases of constrictive pericarditis under discussion, the heart muscle can be regarded as essentially normal. Finally, it is of interest that when improvement or cure is obtained it is maintained. Follow-up studies so far have shown that constriction does not redevelop, at least for the years that these patients have been observed.

## CHRONIC NON-CONSTRICTIVE PERICARDITIS

(Adhesive Pericarditis, Mediastinopericarditis)

The details of the development of chronic non-constrictive pericarditis are meagerly understood. It is fair to assume that the condition is the result of a single or of repeated infection of the pericardium. What is not known is whether recovery in any individual case of acute pericarditis takes place with or without subsequent adhesions. One physician may see a patient with acute pericarditis and twenty-five years later another physician sees the same patient with heart failure. At this time, if chronic pericarditis is present, it rarely is discovered, or if it is correctly diagnosed the past history of acute pericarditis is not known. We have very little reliable data from which to ascertain the frequency with which any one of the primary causes of acute pericarditis produces chronic pericarditis. The complexity of the problem is increased by difficulties in diagnosis. There is reason to suspect that while infection with the tubercle bacillus, the pneumococcus, staphylococcus, and possibly the streptococcus are responsible for chronic



constrictive pericarditis (discussed in the preceding pages) these same infections may account for other cases of chronic pericarditis that are non-constricting. However, when there is also chronic valvular disease with chronic pericarditis all indications are that rheumatic fever is the main etiologic agent. Furthermore, it is of considerable interest that such rheumatic cases never produce the picture of constrictive pericarditis.

From a clinical point of view cases of chronic non-constrictive pericarditis may be divided into two groups—in one the subjects are symptomless and in the other there is significant heart disease and eventually heart failure. In the former group are persons in whom the condition is found at postmortem examination who had shown no symptoms referable to the heart during life. Small bands or synechiae may be found extending between the visceral and parietal pericardium, or at times the entire pericardial cavity may be obliterated and yet there will be no embarrassment to the circulation. Such changes in the pericardium can be seen in patients dying of entirely unrelated conditions such as brain tumor or abdominal cancer.

The second group concerns us here. The affliction in these patients is generally designated as *chronic adhesive pericarditis* or *chronic mediastinopericarditis*. The adhesions in the pericardium are so extensive and firm as to bind the heart to neighboring structures such as the ribs, pleura and diaphragm. Thus the free contractions of the heart are impeded and its work is increased. The condition is found associated with marked cardiac hypertrophy. The adhesions were at one time thought to be the cause of the great hypertrophy. This is probably untrue, for very large hearts with chronic pericarditis also have valvular disease, which alone produces considerable cardiac enlargement. The fact that the pericardial adhesions do account in part, but only in part, for the hypertrophy was shown in a comparative study of the weights of hearts with and without pericardial involvement. The average weight of forty-three hearts with various forms of valvular disease with adhesive pericarditis was 125 grams greater than a group of sixty-two with similar valvular involvement but without pericardial disease.

**Diagnosis.**—The diagnosis of chronic non-constricting pericarditis is difficult, but from a practical point of view is not important. Inasmuch as this type of pericarditis is associated in the main with advanced valvular disease, the physical signs and the symptoms can for the most part result from the cardiac condition irrespective of the pericardial changes. Such patients will have the customary symptoms of valvular disease and, eventually, of heart failure. They will show the signs of some form of rheumatic valvular disease, often aortic, and with it considerable cardiac enlargement. In addition there may be signs (to be discussed in the succeeding paragraphs) that have often been ascribed to the pericardial adhesions. The liver is frequently and the spleen occasionally enlarged. In the type called "Pick's disease" there



may be perisplenitis, perihepatitis, chronic peritonitis and pleuritis. In fact, the affection that used to be called Pick's disease was rather ill-defined and this designation might have been used to include constricting and non-constricting types of pericarditis.

*Signs.*—The so-called "signs" of adhesive pericarditis are very numerous and bear the names of many prominent physicians, but for the most part are unreliable diagnostically. Studies throughout the entire world tell the same tale, *i.e.*, that this condition is generally first recognized at the autopsy table. The main difficulty is that most of the "signs" may be present when the heart is markedly enlarged and the pericardium is normal. Nevertheless, it is well to enumerate some of the signs and to comment upon them briefly.

Systolic retraction of the apex region is a common finding. With systole the heart actually pulls the interspaces inward rather than outward. The heart is also immobile. The apex impulse and left border normally shift an inch or so when the patient is moved from the left lateral to the right lateral position. In chronic adhesive pericarditis the heart is comparatively fixed. On deep inspiration the left border of cardiac dulness may be unaltered because the left lung is prevented from extending over the heart by adhesions between the heart and the anterior chest wall. As a corollary to this phenomenon, on watching both nipples during deep inspiration the left may be found to lag behind or even to remain stationary while the right moves forward (Wenckebach's sign). On examining the interspaces below the angle of the left scapula, a systolic retraction may be seen on careful inspection (Broadbent's sign). A diastolic shock may be felt in the apex region and a sudden diastolic collapse of the jugular veins may be present. A diminution or disappearance of the radial pulse with inspiration (*pulsus paradoxus*) has also been described as a sign of adhesive pericarditis. An electrocardiographic sign has been proposed by Dieuaide. This may be detected by taking electrocardiograms with the patient in the dorsal, left and right lateral positions. Normally the heart shifts under these circumstances, thereby altering the electrical axis. If the pericardial adhesions are sufficient to render the heart immobile, one would expect to find no appreciable change in the electrocardiograms. This test seems to have a sound theoretical basis, but the validity of its practical application is not so certain. I have seen a case in which no shift in the electrical axis occurred by this technique and yet the pericardium was found to be normal.

Finally, the *x*-ray may throw light on the diagnosis. Irregularities in the contour of the heart as a result of adhesions may be seen. With inspiration the heart may fail to descend, as it normally should, if it is adherent to the anterior chest wall, or tugging of the diaphragm or pleura may be detected with each systole on fluoroscopic examination. Other *x*-ray findings, such as diminution of movements of the cardiac



borders or marked calcification (*concretio cordis*), are generally indicative of constrictive pericarditis and have been discussed.

The foregoing long list of signs of adhesive pericarditis is an indication in itself of the difficulty of diagnosis and would lead one to suspect that the signs are not very reliable. In fact, except for the roentgenological evidence, I have seen practically all these findings in patients with enlarged hearts that showed no pericardial disease whatever. I recall a case that seemed quite typical of Pick's disease. The patient was a woman forty-five years old, who showed evidence of advanced cardiac failure. The liver was enlarged, the abdomen was full of fluid and there was a hydrothorax. There were signs that seemed typical of mitral stenosis. In addition there was marked systolic retraction of the apex and immobility of the heart. There was a prominent Broadbent's sign, which was demonstrated to several undergraduate classes in physical diagnosis. The abdomen and chest were tapped many times. The patient was observed very carefully over a period of four years and was always thought to have mitral stenosis, adhesive pericarditis and polyserositis. On postmortem examination, the pericardium was found to be perfectly normal, the heart was enormously enlarged and dilated, and there was stenosis of the mitral, aortic and tricuspid valves. This well illustrates the difficulties in diagnosis.

**Treatment.**—As has been indicated, the importance of accurate diagnosis of chronic pericarditis of the constricting type is great because surgery offers the only effective treatment. This is not true of chronic non-constrictive pericarditis or chronic adhesive pericarditis. For the most part, this condition occurs in patients having rheumatic valvular disease and enlarged hearts. They suffer from cardiac disabilities that are not particularly the result of the adhesions. Such patients would be receiving treatment for heart failure anyway and in most cases the treatment would be no different even if it were known that adhesive pericarditis were present.

Many years ago Brauer devised an operation for the relief of chronic pericarditis which he called "cardiolysis." At that time the essential difference between the constricting and non-constricting aspects of pericarditis were not as well differentiated as they are today. The procedure consisted of removing several ribs and a portion of the sternum over the precordium, thus permitting the heart to tug away at flexible soft tissue rather than at an unyielding bony chest. This operation did not necessitate the exploration of the pericardium and heart. Although carrying much less risk than the Delorme operation, in which the heart, itself, needs to be explored, it is performed very little nowadays. It can do no good for constrictions that may be present. Its only effect would be to diminish the work of the heart in its contraction or to give an enlarged heart a little more room, serving as a decompression of the chest. Inasmuch as the pericardium is not explored, the presence of



pericardial adhesions is not confirmed by the operation. Some patients appear to have been improved by this operation and in a few of these who were subsequently examined postmortem no pericardial adhesions were found. Improvement could then only be ascribed to a decompression effect on an enlarged heart.



## 6

### ANGINA PECTORIS AND CORONARY THROMBOSIS

#### ANGINA PECTORIS

ANGINA PECTORIS is a distinct clinical entity in the sense that when properly understood it can be diagnosed accurately, the subsequent course of the disease can in a great measure be predicted and the type of pathological changes in the heart at autopsy can be foretold. The likelihood of sudden death is a necessary corollary of the term "angina pectoris." In addition, one may expect certain measures that are helpful in some heart conditions to be comparatively useless in angina pectoris and other means not ordinarily employed to be of benefit. If the above is true, then it must follow that the condition is discrete enough to be considered a clinical entity even if there is divergence of opinion as to its underlying cause. It would be a mistake to give up the use of this term in clinical medicine. Pernicious anemia is fairly well understood and generally distinguishable from other forms of anemia although its fundamental cause is somewhat in dispute. Just as it is true that not all cases of anemia are instances of pernicious anemia, nor all sore throats due to diphtheria, so not all cases of chest pain are due to angina pectoris. Likewise, although liver is helpful for pernicious anemia, it is of little use in secondary anemia and whereas antitoxin is specific for a diphtheritic sore throat it is useless for the sore throat of Vincent's angina. Similarly nitroglycerin may relieve an attack of angina and have no influence on the precordial pain of cardiac neurosis or mitral stenosis. It, therefore, becomes imperative to isolate from among those patients who suffer from chest distress the ones who have true angina pectoris.

A patient either has or has not angina pectoris. There is no room in this discussion for terms such as pseudo-angina, false angina, juvenile angina and anginoid. The use of such terms has served to spread confusion and to distort the truth. It may be proper to call a condition



pseudo-angina if the symptomatology is quite characteristic of angina and yet one infers that there is no true anginal element whatever in the problem. Furthermore, the term "mild angina" is often equivocal. If the term means that the complaint is not very troublesome its use is proper, but it must not be inferred necessarily that the disease is mild or trivial. An anginal patient with only mild complaints may be dead of heart disease in a few days, for the underlying process may yet be grave.

Much of the confusion concerning angina has resulted because of the lack of uniformity in the explanations of the mechanism of the attacks that characterize the disease and in the underlying etiology. So divergent have the hypotheses been that one authority has insisted that angina has nothing to do with the heart and is only the result of disease of the aorta, another has maintained that it is due to spasm of the coronary arteries, a third that it is due to heart fatigue and a fourth that it is a neurosis. Although it is not proposed here to discuss the various theories concerning angina pectoris, one thesis recently propounded seems to be worthy of mention. It has been suggested that the invariable mechanism which causes an attack of angina is anoxemia of the myocardium no matter how brought about. This can explain the overwhelming frequency of angina pectoris in disease of the coronary arteries. It also accounts for its occurrence in some cases of pernicious anemia with comparatively normal coronary arteries, for here anoxemia of the heart might well result from lack of hemoglobin. It also accounts for the disappearance of angina in some cases of thyrotoxicosis when the demands on the heart are decreased by diminishing the body metabolism. There are many other features of angina that this hypothesis satisfactorily explains which other theories do not and I feel that at present it is the one most worthy of our consideration.

**Etiological Factors.**—Although there is no single specific organic cause of angina pectoris there are numerous etiological factors which are definitely related to its development. It has generally been known that it occurs in males more frequently than in females, the proportion being about three or four to one. This disproportion becomes all the more impressive when it is appreciated that hypertension is much more frequent in females than in males. It at least throws some doubt on the significance of hypertension *per se* as an etiological factor. Probably the most important etiological factor is heredity. There is a strong familial factor in angina pectoris. All practitioners who have treated members of the same family over long periods of years have noted that there are families with a tendency to sick headaches, others that are neurotic, some with a tendency to asthma and still others with a liability to early death from one or another form of cardiovascular disease. I have seen three brothers all of whom died of acute coronary thrombosis during the sixth decade of their lives. It cannot be mere chance that three members of the same family should die of the same



disease at approximately the same time of life. It seems as if the original tissue or structure that such individuals have at birth must be peculiar, so that ordinary wear and tear does more harm to certain vessels than is the case in other people. It is not altogether too fanciful to conceive that the anatomical configuration of one of the coronary arteries may be slightly different, so that with the repeating contractions of the heart muscle one particular part of the vessels receives undue strain or tension. Even a very slightly exaggerated bend in the course of a vessel could easily account for its premature degeneration. This peculiarity would readily be inherited just as are queer configurations of the lobes of the ears and other structures. It would also explain the decided susceptibility to occlusion of one special portion of the left descending coronary artery, about 2 centimeters from its origin. Another possible explanation of this familial factor of angina pectoris and its predominance in the male sex is that the peculiarities in the structure and function of the endocrine system which we inherit may be the basis of premature arteriosclerosis.

As a corollary of the hereditary influence there is a distinct constitutional type of individual who is particularly susceptible to angina pectoris. I refer to the well-set, strong man who is slightly overweight and who has been especially healthy all his life. So often these patients boast of their previous good health and remark that they "never had a sick day in thirty years," and that they "could do the work of two men." They have been physically stronger than the average, even when their daily activities did not require them to use that strength. The forearms of these individuals are muscular, round and the skin fits snugly over their muscles in contrast to the flat-armed type. It is very striking that angina is quite rare in the undernourished and in those who have been sickly for years. It selects the vigorous rather than the weak.

The average age of patients with angina pectoris when the disease first manifests itself is about fifty-six years for males and about fifty-eight years for females. There are a few young individuals even in the second and third decades with angina who suffer from aortic valvular disease. This relation will be discussed in great detail later. Even apart from these exceptional instances, angina is not extremely rare in the fourth decade and I have even seen an instance of typical angina pectoris followed by acute coronary thrombosis in a young man twenty-four years old, and one in a woman of twenty-two who subsequently went through pregnancy successfully. It certainly seems to be occurring in younger individuals than formerly.

When conditions such as Buerger's disease, hyperthyroidism, and aortic valvular disease, to be discussed below, are not present, young anginal patients between the ages of twenty and forty will generally be found to have other etiological stigmata, such as a marked family history of vascular vulnerability, diabetes, or hypercholesteremia.



It is the opinion of most observers that angina is increasing in prevalence both relatively and absolutely. It is obvious that with the marked increase in the span of life that has taken place during the past century and with the improvement in the methods of diagnosis, there should be a greater number of cases of those diseases that mature in persons in the second half of life. But there is indirect evidence to show that angina is actually more common among a given number of adults than formerly. I am of the opinion that its relative and total incidence has been on the increase and that this increase is real rather than apparent. The reason for this is very obscure. It is generally explained on the basis of the "stress and strain of modern life." This still leaves us in a quandary as to the actual mechanism involved. Has there been some specific deleterious influence at work during these past decades such as the telephone with its terrorizing clang, or the motor car with its noxious fumes, or a dietary defect with an overabundance or lack of some element, or the prevalent habit of smoking? Is it even due to a process of natural selection, those with angina being the more fit and dynamic and propagating their kind, or is it merely the hustle and bustle of the twentieth century? These are important but unanswered questions.

In a very recent study it was found that Jewish patients with coronary artery disease died on the average at a considerably younger age than non-Jewish patients. A similar though less striking difference was found in the age at death of smokers in contrast to non-smokers, the former dying about three years earlier. If this can be confirmed and it can be shown that the overall age of the groups studied were the same as the average age of all patients with angina, it would follow that these two factors are somehow related to the development of coronary disease.

It is of some interest that females are not only less frequently afflicted with angina but that the disease occurs on the average two to three years later than it does in the males. The reason for this is obscure but the facts are beyond dispute. This may be one of the important reasons that the average age of women at death in the latter decades of life as shown by all insurance statistics is consistently three years or more greater than that of men. If the smoking of tobacco has had something to do with this difference in the sexes, as has been thought by some, it can be expected that in the next few decades, as a result of the greater prevalence of smoking among women, the discrepancy between the sexes may disappear or diminish in extent. Arteriosclerosis is much less extensive in the female than in the male sex. This merely puts the problem in different words, for the reason for this lessened susceptibility to sclerosis remains obscure.

Among the specific diseases that are etiologically related to the development of angina pectoris *syphilis* used to be regarded as most important. It was particularly looked upon as a frequent cause of



angina in young people. Since the introduction of the Wassermann reaction it has become evident that syphilis is an uncommon finding in angina. I found it present in only 4 to 5 per cent of the cases and even in these it cannot always be regarded as directly related to the angina. There has been considerable confusion between syphilitic aortitis and angina pectoris. The two conditions are quite different. One need but realize that the former is extremely common in the colored race whereas the latter is very rare, to question any important bearing that syphilis could have in the development of angina. There are occasional instances in which the syphilitic process extends down the ascending aorta and partially or completely occludes the openings of the coronary arteries. In this way anginal distress may result. Furthermore, the development of aortic insufficiency itself with its accompanying low diastolic pressure has been regarded as a cause of angina. I have considerable doubt as to this relationship. However, when there is neither involvement of the coronary orifices or the aortic valves, it is very unlikely that syphilis is responsible for angina, even when both syphilis and angina are present. In a similar way a small number of tuberculous individuals may incidentally have syphilis without any relationship between the two conditions. It is important to bear in mind that syphilis very rarely produces changes in the main course of the coronary arteries where the customary atheromata are so common and that when a syphilitic has such changes it does not follow that the coronary changes are syphilitic or in any way different from those seen in non-syphilitics.

A much more important disease related to angina is *diabetes*. A large number of diabetics, especially the elderly ones with mild cases, eventually develop coronary artery disease. This relationship probably will grow more common as time goes on, for insulin should prevent early death in diabetes and permit such individuals to live long enough to develop the vascular changes that are so prevalent in this disease. It is not altogether certain that diabetes is the actual cause of such angina pectoris as develops, for the age at death of patients with angina pectoris and diabetes is essentially the same as those without diabetes. It may be that diabetes merely indicates the type of individual who has a vascular vulnerability just as the family history and constitutional type reflect those prone to this disease. For the most part it is the mild diabetic fifty or sixty years old, who has not required the use of insulin and in whom the diabetes may be looked upon as one of the evidences of generalized arteriosclerosis, who develops anginal symptoms.

A high cholesterol content in the blood (*hypercholesteremia*) is common in patients with coronary artery disease. This is particularly true of those who have xanthomatosis or even when only isolated xanthelasma lesions are present around the eyelids. It has been maintained by Leary that diets high in cholesterol content produce marked sclerosis of the arteries, especially the coronary arteries, in the experimental animal. Whether this mechanism applies to man and whether it can be altered



by diets that are low in animal sterols or by thyroid administration has not been determined.

Similarly there are other less common diseases that are particularly associated with generalized arterial changes which are etiologically related to angina pectoris. I have reference to *gout*, *chronic lead poisoning*, *Buerger's disease*, *myxedema*, *Paget's disease* and *polycythemia*. In these conditions the development of angina pectoris can readily be visualized as the result of alterations in the coronary arteries similar to changes going on in other vessels of the body. The development of angina in association with Buerger's disease is of especial interest because it accounts for some of the cases of angina occurring in younger individuals in the thirties and early forties.

The presence of aortic insufficiency or stenosis is attended in some cases by typical attacks of angina. This relationship has been explained, but not altogether satisfactorily, by the following mechanism. The coronary arteries are thought by some investigators to be nourished during the diastolic pause of the heart beat, for when the heart is in systole the contracted muscle forcefully occludes the vessels and prevents any flow of blood. During the diastolic relaxation, however, the coronary arteries are open and it is then that the heart muscle is nourished. Aortic insufficiency is frequently accompanied by a low diastolic pressure which results in a diminished nutrition of the heart muscle. It is this relative anoxemia of the heart that is supposed to make it more susceptible to attacks of angina. This mechanism does not explain the exact precipitating causes of the attack for the defect of the aortic valve is permanent and constant, present when the patient is free from attacks. Some other factor is required that sets off the spark to produce a spell of anginal pain. Other possible explanations that bear on the relationship between angina and aortic valvular disease were taken up under the discussion of aortic stenosis (Chapter 4). The relationship of aortic valvular disease and angina explains the occurrence of the latter in a small number of young individuals. There are persons of both sexes in the second and third decades of life with rheumatic aortic valvular disease who have typical attacks of angina. It is important to clearly understand this because although such individuals are liable to sudden death, a characteristic of all cases of angina, they are apt to live many years. The prognosis for duration of life after symptoms develop is generally better than in ordinary cases of angina, because their coronary arteries are essentially normal. They do not as a rule develop coronary thrombosis but rather continue the course of valvular disease and eventually die of congestive heart failure or bacterial endocarditis.

There remain a few other conditions that need consideration. I refer to *severe anemia*, *hyperthyroidism* and *paroxysmal rapid heart action*. If a heart that is otherwise competent and structurally sound is nourished by blood which contains 20 per cent hemoglobin or less as may occur



in pernicious anemia, one can readily see that a state of relative anoxemia may exist in the heart muscle. The same ischemia could result with normal vessels and impoverished blood as occurs with normal blood and narrowed vessels. To be sure, anginal attacks are not common in anemia, probably because there are other compensatory mechanisms that come into play to help nourish the heart and other tissues, such as an increase in the velocity of blood flow and in the minute output of the heart, an increase in the utilization of oxygen and arteriolar and capillary dilation. However, such attacks do take place although it is to be expected that they will occur more frequently when the anemia is marked and when it develops in an individual whose coronary arteries are already showing degenerative changes. It is obvious in these cases that the successful treatment of the anemia may prove helpful for the attacks of angina and may even make them disappear entirely. This aspect of anemia and angina has been generally emphasized, but an opposite clinical relationship has received very little attention. I have seen several instances of pernicious anemia in which cardiovascular accidents developed directly after the red blood-cell count returned to normal. In one such case, as the red blood count went from two to five million in three months, the systolic blood pressure rose from 140 to 240 with the development of hypertensive headaches and a terminal fatal cerebral hemorrhage. Retrospectively, it would have been wiser to diminish the liver therapy and to rest content with a blood count of four million, notwithstanding the slight risk of neurological complications. Similarly, several cases of coronary thrombosis have occurred shortly after the restoration of the red cell count to normal in primary anemia. One, therefore, needs to be on guard for such exceptional complications.

For quite a different physiological reason angina pectoris may result from active hyperthyroidism. Here the mechanism is fairly clear, for a heart that is competent when the bodily demands are normal may find itself embarrassed if the basal demands are increased 50 per cent to 60 per cent. Such an individual may be compared to one who is forced to walk all day long. The heart again is suffering from relative anoxemia because of excessive demands. It is unlikely that a normal heart will be so affected by an elevated metabolism. What is more probable is that the thyrocardiacs who have angina pectoris have some disease of the coronary arteries, which requires an increased demand on the heart to produce embarrassing symptoms. Here also the cure of the hyperthyroidism may eliminate or greatly improve the anginal attacks.

Finally there is a form of angina pectoris that can occur in an otherwise healthy heart as a direct result of a sudden increase in its rate. Any of the forms of paroxysmal rapid heart action discussed in Chapter 13 may be the direct cause of such attacks. Although this relationship is not common, when it does occur it may offer considerable difficulties in interpretation. These transient irregularities may result from an



attack of true coronary thrombosis and yet they can occur in an otherwise healthy heart and resemble acute coronary thrombosis. I saw a man thirty years ago who had severe anginal attacks during bouts of paroxysmal auricular tachycardia when his heart rate would reach 250 and be maintained there for hours and days. He never showed any evidence of organic heart disease, remained in normal health for over twenty years, and only in the past few years has there appeared angina on effort not associated with tachycardia. The probable explanation of the anginal pain under these circumstances is that while the heart rate is extremely rapid the pulse pressure becomes so low that there is very little effective head of pressure to drive the blood around. In the case cited above, the blood pressure for hours and days during attacks was about 96 systolic and 88 diastolic. The coronary artery flow must have been greatly impeded with a resulting anoxemia of the heart. It is evident that such paroxysms of rapid heart action can occur in diseased hearts as well, but that when they occur in normal hearts the prognosis can be excellent, for the arrhythmia is generally within control or at any rate self-limited in its duration.

The above peculiar forms of angina pectoris including those with aortic valvular disease form only a small portion of the entire group, possibly 5 to 10 per cent. All the others have no associated anemia, hyperthyroidism, valvular disease, etc., and represent the type that is commonly met with in general practice. One may ask, what is the basic cause of the attacks in these individuals? There have been endless discussions and numerous theories as to the cause of angina and the final solution has not as yet been reached. The evidence points more and more to the view that anoxemia of cardiac muscle is the most important factor. An observation which supports this theory is the reproduction of anginal attacks in patients suffering from this disease by the inhalation of air containing decreased amounts of oxygen. The theory of aortic origin of anginal pain is losing ground and now most authorities look to the coronary arteries for the underlying defect. I have never yet failed to find some disease of the coronary arteries in any of my own patients with angina that have come to postmortem examination, with the exception of the few who had marked aortic stenosis. It is conceivable that under most unusual circumstances even fatal angina might occur with a structurally normal heart. Men working with nitroglycerin have been known to drop dead instantly on slight effort after being away from their work for a few days. These observations have been made by Cecil K. Drinker and lend support to the theory that the mechanism of coronary spasm is a cause of angina.

To be sure there are patients who pathologically show sclerosis of the coronary arteries in whom angina was not detected in life. This, I believe, does not invalidate the coronary hypothesis as the origin of angina. Even if the coronary view is accepted it is not clear whether the pain is the result of spasm of the coronary vessels, whether it is



due to the dilation of the vessel proximal to the spasm or whether the pain sensation comes from metabolites that are not adequately removed from the area of relative ischemia of the heart. Finally, a most important unresolved question is the precipitating cause of an attack which occurs while the patient is at rest. Organic changes in the heart do not come and go. The coronary artery is no less sclerosed a few minutes before than during a typical attack of angina. What trigger mechanism lies behind the temporary explosion? May it not be the endocrine system, particularly the adrenals or thyroid gland? Some experiments I performed several years ago suggest this hypothesis for it was found that adrenalin almost invariably brought on typical attacks in patients suffering from angina and had no such effects on control individuals. The whole problem of anginal pain although nearing a solution still has many mysteries to be cleared.

**Diagnosis.**—The diagnosis of angina pectoris depends on the proper interpretation of symptoms. For this reason the history is all important. The patient will complain of a peculiar distress in the chest. It is frequently not described as a pain but rather as a disagreeable feeling like "indigestion." It is so frequently associated with the belching of gas that both the patient and the physician are only too prone to regard the condition as abdominal in origin. It is well for the physician to elicit carefully the character and location of this complaint. Too often he regards the complaint as a pain in the precordium when it is neither a pain nor located over the heart. Most commonly the location is in the center of the chest, in the sternum or just to the left of the sternum. Much less frequently it is located over the precordium and very rarely at the apex of the heart. It may arise anywhere from the epigastrium up to the root of the neck and characteristically radiates down the arms, through to the back, the neck, the jaw, the side of the face or to the shoulder. The radiation is more common to the left than to the right, but not infrequently both sides are involved. Occasionally the pain starts peripherally and radiates to the chest and rarely it is only in the arms or back and not in the chest. The discomfort is generally described as a constriction, a feeling of tightness, a burning sensation, a feeling of fulness, a choking, a distress or just an uncomfortable pain. Some will say that their clothes suddenly feel tight and others will state that they want to lift or loosen their shirt. The peculiar terms used by different patients to describe the symptoms are very illuminating and it is well to write down the history in the very terms used by the patient. In this way the physician quickly learns the character of the pain and becomes more proficient in recognizing this peculiar type of distress. Often the complaint is so mild that the patient refuses to call it a pain and resents taking it very seriously. The mild complaint may nevertheless be very ominous.

The manner of development of the distress is also peculiar. It comes fairly suddenly and especially on effort. The activity that is most



likely to bring on a spell is walking. Patients often are able to do a great deal of hard physical work indoors and yet not be able to walk a block or two in the street. The effort of walking up a slight grade is particularly difficult. There are two other factors that accentuate this distress. These are a full meal and cold weather. Frequently patients may be able to walk to their luncheon but have difficulty in returning; or may have attacks when the air is cold or the wind blowing and none when the weather is warm. Chilling only a part of the body by placing ice cubes in the hands can diminish the exercise tolerance of patients with angina so that attacks come more readily. Another peculiarity is that the pain may come at one time after a brief walk and then at other times not recur after a much longer and more difficult walk. Some will complain that they may have an attack during the first or second hole of golf and then no more during the rest of the game, or have a spell going to the train after breakfast and be free the rest of the day. Furthermore, there are peculiar efforts in individual cases that bring on attacks such as shaving in the morning, taking a bath, undressing at night or getting into bed between cold sheets. Only careful history-taking will uncover these variations in symptomatology.

Next in importance to physical effort is mental excitement or agitation. There are many patients who will have an attack on coming into a consultant's office. In this way I have frequently been able to witness in my office the entire development of the anginal spell. Sexual intercourse is another precipitating factor that is not uncommon. A heated argument, suddenly being informed of pleasant or unpleasant news or any form of nervous excitation may be the precipitating cause of attacks. Finally anginal attacks may come without any apparent precipitating cause while the subject is at rest. They may even awake patients from sleep; in such instances we try with only partial success to blame the attacks on dreams. A division has been made by some observers between "angina of effort" and "angina of rest" but I see no useful purpose that can be attained by this distinction. The latter frequently follows the former although occasionally the first attack may be of the form described as angina at rest. There are no important practical differences between the two types except that it has seemed to me that those who have angina and hyperthyroidism are very prone to have attacks at rest.

When the attack comes, the patient is generally brought to a sudden halt. When it occurs in the street he is apt to slowly walk to a shop window, not to attract attention, and wait for the distress to relax. He quickly learns that if he remains stationary for a few minutes the pain disappears and he is able to continue. He also learns that if he does not stop the pain persists and may grow more uncomfortable. In rare instances the attack does not prevent continuation of the effort. During the period of pain in most instances there is no shortness of breath, but some will complain that they have difficulty in breathing. It is hard



to distinguish true dyspnea from a certain immobility of the chest that may accompany the attack. The patient may not want to move his chest even by the act of breathing for fear of aggravating the pain. When true breathlessness occurs with the attack it generally signifies that there is an additional factor of heart muscle insufficiency which for the most part is absent in angina. The occurrence of fear of death is variable. It is often, but by no means always, present. It will depend on the severity of the pain and the psyche of the individual. There is something fearful and ominous about the sensation, however, even although it is not severe. The same patient who dreads an attack of angina may suffer a very violent attack of renal colic requiring repeated hypodermic injections of morphia yet experience no fear.

There are occasional instances in which attacks of angina are related to hypoglycemia. Patients may observe that spells are most likely to occur when they are hungry or may be prevented by eating something sweet. One should particularly suspect this relationship if attacks occur late at night. In these cases blood sugar studies are useful.

There are other cases in which attacks have developed directly after severe trauma to the chest or after a very violent sudden effort. There is now reason to believe that under such circumstances the coronary arteries may be actually traumatized with resultant symptoms of coronary insufficiency. The relationship between trauma and coronary artery disease is a very difficult one to disentangle, because so much depends on subjective complaints which may not be reliable when matters of litigation arise. However, when it is known that an individual was well before such an accident and begins to suffer from definite anginal pain hours or a few days afterwards, it is reasonable to assume that there is a relationship of cause and effect. The same is true when one suffering from simple angina becomes markedly worse or develops evidence of a coronary thrombosis directly after significant trauma.

When a patient is seen during an attack he will appear slightly pale and introspective and there may be slight perspiration. He will not want to converse but will prefer to remain quiet until the attack is over. The pulse rate during this time may be unchanged but more generally is somewhat accelerated. The rhythm remains regular in the vast majority of cases but occasionally extrasystoles may suddenly appear during the attack. Such patients may be helped by quinidine. The blood pressure, if taken during the pain, will rise almost invariably. It is not the pain that produces the rise in pressure in these patients for I have found no rise in these same persons during the pain of severe renal or biliary colic. Occasionally there is an increased flow of urine or of saliva during attacks. After a few minutes, either spontaneously or as a result of a nitroglycerin pill, the distress disappears and the patient feels quite well again.

The preceding details have been gone into because upon their proper evaluation will the diagnosis of angina pectoris depend in the vast



majority of cases. The physical examination and all our laboratory tests are so often of no help that it is only from a true understanding of the nature and character of the symptoms that these cases will be recognized. It is the failure on the part of the doctor to appreciate the importance of mild distressing complaints in the chest that leads many patients with angina pectoris to be overlooked entirely or to be treated for indigestion or gas. It is imperative that we deliberately inquire into the history of all our patients over forty years of age, asking whether they have chest distress on effort no matter what their primary complaints may be, for they may not volunteer this information. The lesson that needs to be repeatedly emphasized is that this common and serious malady is generally associated with no significant abnormal findings on physical examination.

**Physical Examination and Laboratory Tests.**—Although there is very little to be made out on examination that is characteristic of angina one frequently detects some abnormalities in the cardiovascular apparatus. Generally there is evidence of arteriosclerosis in the peripheral vessels or on ophthalmoscopic examination. Often, however, there is no more than would be expected at that age and at times there is even complete absence of any detectable vascular disease. The blood pressure is variable and may be anywhere from subnormal to markedly elevated.

There is a curious and most important difference in the blood pressure findings in the two sexes. Whereas the average readings for males were found to be 149 mm. systolic and 89 mm. diastolic, the corresponding figures for females were 190 mm. and 102 mm. In fact, there were very few instances in a considerable experience in which I found a systolic blood pressure under 140 mm. in a female except a few in whom a previous hypertension had given way to a lower pressure after a coronary thrombosis. The inference is that although normal or low blood pressure is common among males with angina, the absence of hypertension in females, except following a coronary thrombosis, makes one strongly doubt the diagnosis.

The heart in most instances is enlarged although frequently the enlargement cannot be detected without x-ray examination. Occasionally calcification of the coronary arteries can be seen fluoroscopically or on the x-ray film. Although such calcification always means coronary sclerosis, is distinctly pathologic and generally establishes the diagnosis of angina, it occasionally may be found in the absence of angina. It must be remembered that the term "angina pectoris" denotes a physiological state, and sclerosis of coronaries is a structural condition. Although the latter is by far the most important cause of the former, each on occasions may exist without the other.

There is a dominantly regular rhythm in practically all cases. Occasional extrasystoles may be present but auricular fibrillation is rare. There seems to be some antagonism between auricular fibrillation and angina. If the former exists it is unlikely that the latter will subse-



quently develop. The presence of both does occur but is rare. Auricular fibrillation can develop in a patient with angina pectoris following an attack of coronary thrombosis or after angina has been present for some time.

The heart shows no murmur whatever in about one-half of the cases and in the other a systolic murmur is heard at the apex or base of the heart. A small number, possibly 5 to 10 per cent, show a diastolic murmur. This includes the very rare patient (1 per cent) with mitral stenosis that has angina, the young group of rheumatics with aortic valvular disease and a few older patients with aortic insufficiency due to syphilis or hypertension. On the whole the quality of the heart sounds is of no aid in diagnosis. In most instances the sounds are normal in character but in some the intensity of the first heart sound is distinctly diminished. A diminution of the intensity of both heart sounds can be due to a thick chest wall or overlying emphysematous lungs and the like, but if the first sound is muffled and the second sound is distinct, it at least makes one suspect that the heart muscle is diseased. In some a gallop rhythm may be heard. There is no evidence of congestion in the liver, lungs or extremities unless there is an added element of congestive heart failure which will be found in a comparatively small number.

There are two other examinations commonly carried out in studying patients with heart disease, *i.e.*, determination of the vital capacity of the lungs and electrocardiography. The *vital capacity* is the greatest amount of air one can expire after inspiring as much as possible. This varies mainly with the height of the body. Normal standards have been established and whereas formerly they were calculated with reference to given heights this was later changed and comparisons were made with reference to the surface area of the body. In this latter calculation the surface area is determined from the height and weight of the body. This change in calculation seemed more scientific and accurate when it was first introduced, but I have always felt that the reverse is true. One can obtain a closer reading of the expected or normal vital capacity from the height than from the surface area of an individual. The following example will make this clear. A young man twenty-five years old, in good health, weighs 135 pounds and is 67 inches in height. His vital capacity is 4000 c.c., which is perfectly normal. In ten years he gains 40 pounds, is still in perfect health, the vital capacity is still 4000 c.c. and his height is the same. The surface area has increased and the "expected or normal" vital capacity has increased. Calculating on the basis of height his vital capacity is 100 per cent of normal, while in terms of surface area it has fallen to about 80 per cent. This gives one a false impression, for his circulation has been normal throughout. There is no reason why one should expect a greater vital capacity because the subject has become obese. In fact the reverse occurs when obesity is marked. A patient may have no disease of the cardiovascular



system and yet have a distinct diminution in the vital capacity of the lungs from obesity alone. When the added weight is sufficient, it may inhibit the expansion of the chest and the descent of the diaphragm. This often results in dyspnea on effort. This type of diminished vital capacity and shortness of breath does not indicate heart disease, is not of itself progressive and must be carefully distinguished from the other more serious type of breathlessness. This may entirely disappear with appropriate dieting and a consequent loss of weight. A corollary that follows from the foregoing is that, *ceteris paribus*, a thin cardiac who is short of breath is apt to be in a worse state than an obese cardiac, for, in the case of the latter, part of the dyspnea at least is due to the benign obesity. Similarly, a low vital capacity reading is more ominous in a thin than in a stout individual. It is apparent that the readings must be interpreted in the light of the constitutional type or physique of the individual.

Age likewise influences the vital capacity to some extent. Practically all the normal standards with which readings are compared have been obtained from young men and women, generally college students or young hospital interns and nurses. There are no extensive figures for normal older people. The result is that the vital capacity of patients with angina pectoris is being compared to normal young people. The readings thus obtained would indicate that in angina the vital capacity is diminished about 20 per cent to 25 per cent. If suitable comparisons could be made this difference would be much less and probably would show that when unassociated with myocardial insufficiency the vital capacity in angina is essentially normal. What has just been stated about the vital capacity in relation to age and weight needs to be borne in mind in interpreting readings. It is difficult to compare the reading in one patient with that of another unless the differences are marked, and yet smaller changes occurring in the same individual from time to time may have more definite significance. This is particularly true in following the same patient over the course of years.

In the interpretation of the relation of the vital capacity of the lungs to breathlessness, two other factors need consideration. The first is the general muscular strength or tone of the body, especially the muscles of respiration. With marked anemia or with general asthenia, although the total breathing space is normal, the effort of taking deep breaths rapidly is exhausting and cannot be kept up for any length of time and breathlessness results. The second is the speed with which the respiratory cycle can be completed. In marked emphysema, expiration is so difficult and prolonged that the number of respiratory cycles per minute is considerably reduced. In the last analysis it is not only the actual vital capacity of the lungs but the facility with which a given amount of air can be inspired per minute that determines the development of dyspnea.

The *electrocardiographic findings* in angina pectoris are variable.



Frequently they are perfectly normal or they show preponderant hypertrophy of the left ventricle which has no diagnostic significance. On the other hand, in a good many cases there will be distinct abnormalities of the ventricular complex. Not all of these changes are particularly indicative of angina. In fact some of these are also found in association with heart disease other than angina, but in so far as they direct attention to a pathological condition of the heart they aid in avoiding the error of making a diagnosis of normal heart. Interest has recently developed concerning the presence of a prominent Q wave in Lead III. Although such a wave is found not infrequently in a person with a normal heart it has appeared to be much more common in those suffering from coronary artery disease. There are other changes that point more clearly to angina pectoris inasmuch as they are so commonly associated with disease of the coronary arteries. Among these are spread and delay in the Q-R-S complex, bundle branch block and initial ventricular complexes of low amplitude. All such alterations occur in heart disease other than angina but for the most part are not found in normal hearts. Other abnormalities that more clearly point to coronary artery disease are certain changes in the Q-T interval or in the T wave. These changes will be discussed in greater detail under coronary thrombosis but in so far as some of them are observed in cases of angina they often are diagnostic. Occasionally opportunities have been afforded for obtaining electrocardiograms of patients during an attack of angina and some have shown slight but distinct alterations of the R-T interval resembling those seen in coronary thrombosis. The R-T segment is somewhat depressed or elevated in one or another lead as compared to the form that is seen in the tracings before or after the attack. This has been regarded as additional evidence to support the theory that anginal attacks are due to myocardial ischemia. The importance of electrocardiography is that, although often it is of no help, in some doubtful cases of angina the tracings may be the only evidence of the grave condition that exists and that without such evidence the patients would be regarded as having a normal heart.

When the diagnosis of angina is still in doubt there are some tests that may prove helpful. The first is to have the patient exercise with the purpose of bringing on the distress of which he complains. This can be done by making him walk upstairs until pain is produced. One might still be in doubt whether the type of chest pain thereby brought on is anginal or not. Another test is to have the patient breathe an atmosphere poor in oxygen content (10 per cent). If this is continued for ten to twenty minutes, anginal attacks may be reproduced in those suffering from this disease. Furthermore, breathing low concentrations of oxygen may produce abnormalities in the R-T segment and T wave which do not occur in normal hearts. Somewhat similar electrocardiographic changes may be produced by a brief effort. When these are marked such as shown in Figure 116 the diagnosis of coronary



sclerosis is quite clear. Finally, I have employed an adrenalin test for angina that merits a word of explanation. It was found that the subcutaneous injection of 0.5 c.c. to 1 c.c. of 1 : 1000 solution of adrenalin reproduced attacks of angina pectoris in most cases and failed to do so in control cases. In some patients even smaller doses were effective. Unfortunately adrenalin injections are dangerous when given to patients who have heart disease and especially angina. This test, therefore, should never be employed when the diagnosis is quite definite and the amount employed in performing the test should be small at first (*e.g.*, 0.3 c.c.), increasing the amount if necessary on subsequent examinations. When positive, the patient will start complaining of the same sort of pain in the chest five to fifteen minutes after the injection is given. The pain will generally be accompanied by a rise in pulse rate and in blood pressure. Just as soon as the pain is reproduced, nitroglycerin, amyl nitrite or, if necessary, morphine is given to bring the attack to an end. I believe that the test can be helpful in some doubtful cases, particularly when it is felt that the condition is functional rather than organic.

There are several conditions that particularly must not be confused with angina. First, there is the large group of patients suffering from some form of organic heart disease, valvular or hypertensive, who complain of pain in the chest. For the most part the pain is at the apex, not in the sternum, and is often associated with hyperesthesia of the left breast. Then there is the group who have functional heart disease. Here the pain is also apical and generally comes at rest. Furthermore, arthritis of the spine, subdeltoid bursitis and other muscular pains from trauma and the like may simulate angina. The confusion becomes increased because many patients with coronary disease develop a troublesome pain in the left shoulder, arm and hand that resembles causalgia and is not due to the heart. They may, therefore, have two different types of pain in the left arm, one of an anginal type brought on by effort and another related to motion of the left arm or shoulder. The two need to be carefully distinguished, for the significance and treatment is quite different according to the type that is present. For the shoulder pain local heat and aspirin may be all that is required. Quite recently disabilities in the hands have been reported as associated with coronary artery disease, especially following myocardial infarction. Dupuytren's contraction and stiffness of the fingers resembling sclerodactylia presumably due to vascular constriction have been observed. A most important and common differential diagnosis is that between angina and gallstones. The difficulty is increased because so many have both conditions. x-Ray of the gallbladder is of great value in this regard. Other conditions, such as diaphragmatic hernia, herpes zoster, cervical rib, thoracic tumor or aneurysm, may also cause confusion. A complete review of those features that characterize angina pectoris, however, will generally lead to the correct diagnosis.



Despite all our routine methods of study many patients with angina will show either nothing abnormal or there will be found only those minor alterations that are common in otherwise well people in the second half of life. Even when abnormalities are found they do not always characterize the anginal state. The description of the type of distress is by far the most important feature from a diagnostic point of view. Despite the great dependence on the subjective symptoms and notwithstanding the fact that they may be atypical and simulated by diseases other than angina, the diagnosis of angina pectoris by and large can be made with a high degree of accuracy.

**Prognosis.**—The prognosis in any case of angina pectoris is most uncertain. Sudden and unexpected death which characterizes this disease may occur at any time. It probably is just as well for all concerned that accurate predictions as to this fatal outcome cannot be made. There is no other condition in which sudden and instant fatalities take place with the same degree of frequency. In fact, when the diagnosis of angina pectoris is properly made the possibility and likelihood of this eventuality is always inferred. A word about instant death may be appropriate at this point. There are very few conditions that cause instant death. Physicians are too prone to make the diagnosis of cerebral hemorrhage, stroke or apoplexy when a patient dies suddenly. In my experience a rupture of a cerebral vessel from hypertension or cerebral aneurysm, or a cerebral embolism never kills instantly. Such patients may suddenly become paralyzed or comatose but death, when it occurs, follows after several hours or more commonly in a few days. Major peripheral emboli do not kill instantly. Even a large pulmonary embolus presents a picture of sudden respiratory distress and suffocation and when fatal, death is apt to be delayed for a period of ten minutes or more. Occasionally rupture of an aortic aneurysm may produce an instant fatality. In fact most of the cases in which death occurs instantly, *i.e.*, within seconds or one or two minutes, are directly due to the heart and moreover are the result of a limited number of causes.

Instant death may result from rupture of the ventricle. This is by no means an uncommon event in those suffering from coronary heart disease and is also an occasional end-result in subacute bacterial endocarditis. Another cause is complete heart block. Here the circulation may be quite adequate and compatible with a considerable degree of physical activity as long as the ventricles contract at a rate of 30 or more. When an attack occurs in which there is failure of the ventricle to respond (Adams-Stokes' seizure) unconsciousness results and if the asystole lasts more than a few minutes consciousness does not return and the attack is fatal. There is a very rare condition in which inhibition of the heart occurs. It is not a matter of blocking of heart beats but rather that the beats do not arise; the pacemaker stops functioning. This can be due to an oversensitive carotid sinus. It is known that complete arrest of the heart may result for as long as even ten seconds by



this mechanism and it is conceivable that rarely a fatality may occur. The constant use of ephedrine in patients who present this peculiarity is of considerable value in preventing attacks. Sudden fatalities occasionally occur during the administration of quinidine. There have been a few observations that throw some light on this, in which it was found that occasionally quinidine causes inhibition or paralysis of the auricles. Such cases have shown a temporary disappearance of the auricular complexes (P waves). If this same inhibition which has been shown to affect the auricles should affect the ventricles, the heart beat would suddenly stop. In this way sudden death during quinidine administrations, not the result of other causes, may be explained. A similar mechanism rarely follows excessive digitalis (Chapter 21, Figs. 14, 15).

The last type of disorder that causes instant arrest of the circulation is ventricular fibrillation. This must not be confused with fibrillation of the auricles. In the latter condition the ventricles are contracting irregularly but actually expelling blood into the circulation. In the former the ventricles are essentially immobile, producing no effective systole whatever and no flow of blood. There are many reasons to believe that this mechanism is probably an important cause of sudden death. In animal experimentation the heart is frequently seen to stop as a result of the onset of ventricular fibrillation after ligation of one of the coronary arteries. This has generally been considered as the cause of the sudden type of death that characterizes angina pectoris. Besides the above indirect experimental evidence in support of this explanation, there has been recently more direct proof of this point of view. During some routine work it so happened that a patient suffering from angina pectoris was having an electrocardiogram taken. He died in the attack and postmortem examination showed disease of the coronary arteries but no acute occlusion. The electrocardiograms were typical of ventricular fibrillation. This was direct proof that instant and unexpected death in angina pectoris could be brought on by ventricular fibrillation. This mechanism is very likely a common cause of sudden death accompanying coronary artery disease.

Because of the unpredictable occurrence of sudden death in cases of angina pectoris the prognosis in any given instance is very uncertain. Patients may die in the first attack or shortly after the onset of the first symptoms or may live in comparatively good health for over twenty years. There is very little in the clinical features that enables the physician to distinguish the one type from the other. The average length of life after the first symptoms have developed is about four and one-half years for both sexes and the average age at death is sixty-one years for the males and sixty-three years for the females. A more recent review by White has given about seven years as the average length of life after the first symptom. This increase, to a large extent, may be explained by more careful history taking. If, on accurate questioning,



an earlier date of onset of symptoms is elicited, the duration of angina will obviously be lengthened. It was found that whether the tonsils had previously been removed or not made no difference in the course of the disease. I believe that foci of infection have no important relation to this problem. Slight differences in average life expectancy may be predicted on the basis of certain factors but on the whole such considerations do not help materially in estimating prognosis. When angina develops before the age of fifty years the duration of the disease is about two years longer than when it first begins in later years. Those who have a low blood pressure develop the disease a few years earlier and live a little longer after the onset of the disease than those with hypertension.

There is one significant factor that aids in prognosis, *i.e.*, the hereditary one. Those patients with angina pectoris whose parents died at an older age lived distinctly longer than those whose parents died at a younger age. In this study of heredity it was also found that although males suffered from angina about four times as frequently as females, the inherited defect of vascular vulnerability was transmitted more prominently through the mothers of these patients than through their fathers. In other words the females transmit this tendency more than do the males but are themselves less affected. This I believe helps to explain the fact that in the latter decades of life, all vital statistics show that females outlive males by more than three years.

The relation between angina pectoris and cardiac decompensation is of some interest. Although it is generally stated that the former disappears when the latter develops, this is frequently not the case. In fact there are instances in which both conditions are present simultaneously and the disappearance of the evidences of decompensation is accompanied by the disappearance of anginal attacks. The presence of hypertension makes it more likely that congestive heart failure will develop sometime during the course of the angina. The duration of life, however, after the onset of angina is one year longer in those who decompensate than in those who do not. The great frequency of hypertension in women, therefore, explains the more common occurrence of cardiac decompensation in this sex when angina has been present.

Obesity was not found to affect either the age of onset or the duration of angina pectoris. I believe that if obesity *per se* acted deleteriously as a causative or aggravating factor the obese patients should have developed the disease earlier in life and died at a younger age than those with normal or subnormal weights. The fact that this was not found to be true suggests that obesity has no important direct relationship to angina pectoris but merely reflects the constitutional type that is more prone to the disease.

Although electrocardiography is often of considerable value in the diagnosis of coronary artery disease its aid in prognosis is very slight. In a study of over 100 cases in which electrocardiograms were taken



it was found that the duration of the disease was one year longer in those with normal curves than the average, but inasmuch as angina developed earlier in life in the former than in the latter, these patients died at an earlier age than those with abnormal electrocardiograms. The duration of life after the onset of angina was one year less in those with inverted T waves in Leads I or II than in those without such changes. In a small number of instances showing other electrocardiographic changes, such as prolongation of the P-R interval or the Q-R-S complex or Q-R-S waves of low amplitude, the prognosis was slightly better than for the entire series. It must be recalled that in many of the cases with these latter electrocardiographic abnormalities, the anginal features had become less prominent and the element of myocardial insufficiency with dyspnea and limitation in physical activities had become more important.

It is of some interest to know what type of exitus is to be expected in patients with angina. Approximately 50 per cent were found to die suddenly. In addition about 30 per cent died of coronary thrombosis. In these latter cases the patients were seen and lived long enough for the physician to make this diagnosis. Many of the instant deaths in the former group no doubt were also due to coronary thrombosis. That this is not invariably true is attested by the fact that in some cases of sudden death from angina pectoris although atheromatous changes were noted in the coronary arteries there was no evidence of an acute thrombosis. Somewhat less than 10 per cent of the subjects died of congestive heart failure and the remainder died of miscellaneous causes such as cerebral hemorrhage, bronchopneumonia, cancer and other conditions. The fact that there was such a high incidence of that type of death which one would expect in cases of angina pectoris is valid proof of the great accuracy of the clinical diagnosis.

**Treatment.**—The treatment of angina pectoris may be divided into two problems, the treatment of the individual attacks and the general care of the patient with the hope of diminishing the severity or frequency of recurring attacks. Patients with angina should be instructed concerning the use of nitroglycerin. This is the simplest and on the whole the most useful medication for the attack. They should always carry some pills with them. The dose should be  $\frac{1}{100}$  grain or  $\frac{1}{200}$  grain (0.6 milligram or 0.3 milligram). Although most patients find  $\frac{1}{100}$  grain to be the suitable dose some will find either  $\frac{1}{200}$  grain or even  $\frac{1}{400}$  grain to be sufficient. When nitroglycerin is first given it is wise to start with the smaller dose as occasionally the ill-effects from the full dose so alarm the patients that they refuse to use it any more. The pill should be placed under the tongue and should be dissolved in ten to twenty seconds. This should occur either spontaneously or as the result of sucking the tablet or crushing it between the teeth. Tablets that are hard and dry and require minutes for complete dissolution should not be used. The occurrence of a sensation of warmth or pounding



in the head serves as a guide as to the potency of the pill. The patient should be told to use it whenever he has the anginal distress unless it is so mild and of such short duration that it would be hard to know whether nitroglycerin really did much good. Those suffering from angina generally learn before long whether the pill is helpful or not. If its use appreciably diminishes either the duration or the severity of the attack it should be used. The physician should further advise that nitroglycerin is to be used to prevent or anticipate attacks. During the daily routine patients often learn that a particular act or set of circumstances frequently precipitates attacks. Shaving in the morning, undressing at night, walking to the train, attending a business conference, the act of sexual intercourse, etc., are common precipitating factors and nitroglycerin can serve as a preventive in anticipation of such attacks. Occasionally patients may find that taking a pill every two hours during the day may be helpful. I have seen no ill-effects resulting from the liberal use of nitroglycerin when it seemed indicated. One woman consumed 1000 pills ( $\frac{1}{100}$  grain) in one week without ill-effects and there are many patients who have used 100 pills a week continuously for many years.

It is very rare that amyl nitrite is needed or will accomplish what nitroglycerin will not. The inhalation of amyl nitrite acts somewhat more quickly and more violently but is not as simple for daily routine use. On the whole amyl nitrite is used very little. Alcohol in the form of suitable drinks is often of considerable help either during a spell or better still as a preventive. Some individuals find  $\frac{1}{2}$  to 1 ounce of whisky taken before bedtime will prevent attacks from coming at night. Finally the patient should either stop the activity that brings on the spell or he should at least slacken his pace. Although it is well to give this advice it is generally unnecessary as sufferers from angina quickly learn that if they carry on and do not heed the distress it will continue or grow worse and if they stop still the pain lets up. Some attacks are severe and protracted enough to require morphine. Occasionally attacks are instantly relieved by carotid sinus pressure. I have seen many instances where pain disappeared as quickly as two to three seconds after massaging the carotid sinus. Whether such an effect is a neurogenic phenomenon or the result of slowing the heart is uncertain. It throws some doubt on the anoxemia theory of anginal pain, as the pain has been observed to actually disappear while the heart was not contracting. The relief of pain seems to be too instant (in two seconds) for the effect to be chemical. Toxic metabolites require a longer time for their production and their action; likewise, the circulation of blood through the coronary arteries and therefore the new supply of oxygen to the myocardium are very likely less rather than more when the heart stops for a few seconds. These observations on the cessation of anginal pain as a result of carotid sinus slowing are more in accord with the theory of neurogenic spasm.



The second aspect of the treatment is much more difficult and less effective. In most cases the transient attacks are readily controlled by the measures just mentioned. But how are we to diminish the recurrence of these attacks so that they will not prevent the patients from walking or working or conversing? There is no simple specific procedure that has proved very valuable for this purpose. At the outset the question arises whether or not to enforce a period of strict rest in bed. There have been two diametrically opposite views in relation to this method of treatment. In one the importance of a period of bed rest for as long as several weeks or even months has been propounded. In the other it has been maintained that this is not desirable, but it has been recommended that the patient remain ambulatory and walk or exercise to the limit of his tolerance, *i.e.*, he should avoid the effort that brings on the distress if possible. The very fact that such different views have been expressed and carried into practice by equally learned clinicians must mean that neither one can be universally applied nor that there can be any great difference in the results obtained. Many considerations must be taken into account, especially economic ones. One hesitates to put a man to bed for four weeks when the loss of financial income that would result would work great hardships on the family. Particularly would this be true if such a procedure would jeopardize the patient's job or subsequent work. Working men and women over fifty years of age are readily discharged by their employers only to have their places filled by younger employees and find it extremely difficult to replace themselves in industry. These are matters that the physician must constantly keep in mind, for too often the social and economic status of patients is ruined without any significant gain in their health to compensate for this preventable loss. On the other hand, when the person in question has no occupation, is retired from business or has sufficient means to enjoy the luxuries of life one would be more ready to try a period of prolonged rest.

When attacks are recurring very frequently and are quite severe or when they actually prevent a person from attending to his work it is wise to try a period of bed rest for several weeks. In some cases this alone improves the condition or at least renders the patient free from attacks or diminishes the frequency for a considerable period of time. Although the rigidity of the rest treatment will vary with different circumstances in so far as possible it should provide complete mental and physical relaxation. When kept at rest such patients should be allowed to leave their beds only for movements of the bowels. They should avoid all annoying experiences, such as disturbing visitors and business conversations. If there are pressing personal or business matters that are preying on the patient's mind which could be straightened out by a visit of a few hours at the office or by a conference, it is better to have it over with before instituting the rest treatment. In other words all reasonable measures should be employed to procure the rest that



is desired. It is generally better to do this in the patient's home or better still in a hospital than to send him off for a vacation, a procedure that is often ill-timed, expensive and ineffective.

Another consideration in this treatment is the diet. Many patients with angina pectoris are overweight and it will be found that when a period of bed rest is instituted, the semistarvation milk diet (Karell diet) will often prove very valuable. This consists of 200 c.c. of milk four times a day and no other food, allowing a little more water for thirst. This is a very low calory diet and if continued for three days is apt to result in a feeling of weakness and hunger. Accompanying this asthenic state attacks of angina which were occurring daily at rest, may disappear. After a varying number of days of the Karell diet, small additional quantities of other food are gradually added. Five small meals a day are preferable to three larger meals. A high carbohydrate diet has been recommended by many and even the intravenous injection of concentrated glucose solution is practiced by some. I have seen too little good come from the latter to advise its use although I frequently suggest the taking of sweets in the absence of diabetes. When attacks of angina are related to hypoglycemia a high protein rather than a high carbohydrate diet is advisable, for this will insure a more even amount of sugar available in the blood stream. Extra small feedings between meals also help to prevent the fall in blood sugar that is responsible for the attacks. It is important that the diet contain the necessary vitamins and that it should be such that the patient does not gain weight. It is well to advise a period of rest directly after meals whenever possible or at least to urge against any avoidable physical or mental effort on a full stomach, as attacks are very common under these circumstances. In so far as they can, patients should avoid whatever they have learned is apt to bring on attacks.

Whether the patient has been put to bed or remains ambulatory, a loss of weight is desirable in most cases. When the amount of overweight is considerable I have seen marked improvement result from a gradual loss of 30 to 40 pounds in instances where nothing else was done, the patient remaining at work and taking no medication. When the weight is normal at the start, a slight loss of weight might be advised.

The relation of "gas in stomach" and anginal attacks has always been puzzling. A great many patients are certain of the association so that they think they have stomach trouble or "indigestion" and are never convinced that they have anything wrong with the heart. They feel that gas brings on the attack and if they raise the gas the attack stops. It is possible that gas is swallowed before or during the attack and later expelled. On close examination it generally becomes clear that gas may be an accompaniment or result of the attack but that some physical or mental effort is the real precipitating cause and that gas with the patient at rest produces no distress. Notwithstanding this,



some would be a good deal more comfortable if the gastro-intestinal tract were improved. This is no simple matter and taxes our ingenuity to the utmost. Proper bowel habits and whatever measures that will prevent constipation and straining at stool are indicated.

One should not forget that peptic ulcer is by no means rare and disease of the gallbladder with or without stones is very common in patients suffering from angina. When these additional diseases are present their symptoms must and can be distinguished in most instances from those resulting from the heart disease, and merit appropriate treatment. It must not be expected that removing the gallstones will cure the patient of angina. When this is supposed to have occurred I feel certain that the diagnosis of angina was incorrect. I have had a great many patients with both conditions and found that when the gallbladder and stones had been removed surgically, those symptoms due to biliary disease disappeared but those due to the coronary artery disease persisted although they may have been ameliorated.

When the question of smoking came up in former years I used to tell patients to smoke moderately, *i.e.*, not more than eight cigarettes or two cigars daily. Now I am more inclined to urge omitting tobacco entirely. It is generally believed that tobacco has vasoconstricting effects on the peripheral arteries. It should always be avoided in a condition like Buerger's disease. We now know that tobacco produces temporary depression of the T waves in the electrocardiograms in some individuals. In fact marked electrocardiographic changes characteristic of extensive myocardial infarction have been observed to appear shortly after smoking and entirely disappear within fifteen minutes. This makes one think that a major coronary artery was temporarily in spasm producing local ischemia. Although I have never seen tobacco *per se* produce angina, in the sense that the disease would disappear on omitting its use, one does find patients who have fewer attacks if they do not smoke. It therefore seems wise to greatly curtail or to eliminate its use in cases of angina. One should individualize in this matter. One might wisely urge a man, forty years old who has been smoking 40 cigarettes a day to give up smoking entirely and yet permit a man seventy years old to smoke a pipe or a cigar after each meal. In the first instance there is good possibility that the patient may live many years, while with the older individual the number of pleasurable experiences may have already become quite limited.

A variety of drugs has been used with the purpose of diminishing the frequency and severity of attacks and of enabling the patient to do more work without discomfort. The great diversification of these drugs is ample evidence of their inefficacy. Potassium iodide (10 drops t. i. d.) has long been used in all forms of vascular disease including angina pectoris. The frequent reference to the value of this drug that is so prevalent in the older medical literature no doubt is due to the striking results that were obtained in some cases of masked hyper-



thyroidism and in syphilis when their etiological significance was not understood. Whether it is of any value in other cases is problematic and at least doubtful. Digitalis has been used a good deal and it has been found that when angina is unassociated with dyspnea, general myocardial insufficiency or congestive failure it has been of no use. There are some who believe it can aggravate angina pectoris. It should be tried only when there is dyspnea apart from the anginal pain or when there is definite or suspicious evidence of congestive failure.

The use of atropine was once advised by Sir Clifford Allbutt on the basis that the sudden fatal outcome that occurs in angina is due to "vagal inhibition of the heart." There has been no proof either of this mechanism of death in angina or that atropine acts beneficially. Inasmuch as some believe that an increase in heart rate is an important factor in the development of attacks and atropine can increase the heart rate by diminishing vagal tone the therapeutic indications for this drug are at least open to question.

The common experience that all physicians have had, in finding that emotional factors seem to precipitate attacks of angina has led to the use of sedatives. Sir James Mackenzie believed that more good was to be obtained from bromides than from any other medication. The purpose is to diminish the nervous receptivity or instability of the patient so that those reflexes that are involved in the production of the attack or in the sensation of the discomfort will be dampened. For this purpose 1 gram (15 grains) of sodium bromide or 0.015 to 0.03 grain ( $\frac{1}{4}$  to  $\frac{1}{2}$  grain) of phenobarbital may be used three times a day. I have witnessed occasional instances in which patients who were having frequent short attacks at rest have become entirely free from attacks promptly following such medication. Unfortunately this is not the usual result. At times it is distinctly valuable to render the patient semiconscious for a day or two to break up a storm of recurrent attacks of angina using a preparation like sodium amytal 0.2 gram (3 grains) every few hours. In general I believe that the use of some form of sedatives, especially bromides, has a distinct place in the treatment of those patients who have frequent attacks of angina.

Because of the importance of the psychic and nervous factor it is best for the physician to avoid the term "angina pectoris" in talking to his patients whenever possible. It carries too great a dread in the mind of the average lay person today. When it is necessary to give explanations it is less shocking to state that the arteries within the heart are not as flexible or patent as normal or that the heart is simply tired.

A sojourn to a warm climate is often advisable when circumstances permit this luxury. It is a common experience that patients who can not walk a block during the winter in New England without pain are able to walk long distances in Florida. Not only is it warmer but the weight of the clothes worn is less and the individuals are more relaxed.



During recent years there has come into vogue a series of new preparations that are supposed to dilate the coronary arteries and increase the blood flow through the heart. There is considerable experimental evidence in animals to show that these drugs do increase coronary flow. Whether similar effects are produced in human beings and especially in those who already have atheromatous changes in the coronary arteries is another matter and more difficult to prove. There is clinical evidence, however, although not all are agreed on this point, that some patients are more comfortable and can do more while taking these drugs. Of this group the common ones employed are diuretin (0.3 to 0.5 gram t. i. d.), aminophyllin (0.1 to 0.2 gram, t. i. d.), phyllisin (0.25 gram, t. i. d.), quinidine sulfate (0.2 to 0.3 gram, t. i. d.), theocalcin (0.3 gram, t. i. d.), theominal (0.3 gram, t. i. d.), theobrominal (0.3 gram, t. i. d.), erythrol tetranitrate (0.03 to 0.06 gram, t. i. d.), theobromine sodium acetate (thesodate) (0.5 gram, t. i. d.), and others. Physicians should employ those preparations that are least expensive and should omit them entirely if their observations indicate that no clinical improvement takes place. It is well to try patients on such a preparation for one month, then omitting it for a month, alternating in such fashion and then deciding whether attacks are more or less troublesome on one regimen or another. One unexpected complication has been occasionally observed following the use of aminophyllin, *i.e.*, the precipitation of gout. Whether this compound breaks down to uric acid in the body or not is not clear.

Intramuscular injections of various tissue extracts have also been tried, but there is little valid evidence of their value. Cobra venom injections have been employed, with some success, in stubborn cases when attacks were recurring frequently. Although numerous other drugs have been used at one time or another they have generally proved disappointing. These few which have just been discussed, however, are worth a trial but great expectations should not be entertained from their administration. Of these aminophyllin, theobromine and their allied preparations and quinidine are the most promising.

As happens in any chronic disease for which no specific effective treatment is available, many bizarre therapeutic methods are suggested and often supported enthusiastically by their respective sponsors. *x*-Ray treatment over the adrenals has been recommended on the basis that epinephrine acts as the trigger mechanism responsible for attacks of angina. Now testosterone propionate given intramuscularly has been heralded as a valuable cure. I have tried all these medications and have failed to be convinced that they have any real specific value. Finally it is thought by some that a snug abdominal belt applied so that pressure is exerted upwards elevating the diaphragm can prevent attacks of angina on effort. This type of belt does help breathing in cases of emphysema and possibly in cardiacs with congestive failure and also makes patients with enlarged liver more comfortable. It,



therefore, may indirectly be of some value in selected cases of angina and deserve further consideration.

**Surgical Procedures.**—The recognition of the limitations of ordinary medical treatment for many diseases, the lack of specific therapy and the rapid development of surgical technique and newer surgical procedures has brought the general surgeon into contact with problems that not so long ago were entirely confined to the attention of the physician. This has been true of practically all the organs of the body. The most recent spheres of surgical interest have been the lungs, the brain and last of all the heart. We are now seeking surgical aid in the treatment of cardiac disease because of the failure to give our patients relief using the older methods. The problem of heart disease in this respect is difficult for many reasons. The morbid processes are for the most part progressive. The fundamental cause that is responsible for these changes is generally poorly understood so that further progress of the processes can be altered but very little. Finally the obvious technical difficulty of surgical manipulation of the interior of the heart has impeded the kind of therapeutic progress that has taken place in the treatment of disease of the kidneys, lungs and other organs. Despite all this, attempts which at first might have appeared to be desperate have been made. Early in this development simple traumatic wounds of the heart were repaired. Some forty years ago Brauer introduced the operation of cardiolysis for adherent pericardium. This formed the stepping stone for further surgical work that has proved of real value in some otherwise hopeless cases of pericardial disease. Some years ago Cutler and Levine reported the first case of valvulotomy for mitral stenosis. Although the first patient survived the operation and seemed to be somewhat improved, the great operative mortality of the procedure at present renders it impracticable. Likewise the surgeons have been playing their role in attempts to relieve anginal pain.

The first surgical attempts to relieve attacks of angina pectoris consisted in removing the cervical sympathetic ganglia. This was first performed by Jonnesco following the suggestion made many years ago by the physiologist François Franck. At first the entire chain consisting of the superior, middle, and inferior ganglia, including the stellate ganglia generally on the left side, was removed. Later a simpler operation in which only the superior ganglion was removed was proposed by Coffey and Brown. The results obtained by these operations were rather variable. It seemed that in about one-half of the cases some relief was obtained but it was impossible to predict who would and who would not be improved. I have had some patients that were greatly helped by this simple operation, one who was enabled to walk miles without distress, and yet there were others who seemed equally promising subjects who were hardly relieved at all. There was much controversial discussion concerning the rationale of these operations



as the sensory and motor pathways to and from the human heart were not well understood. The most recent point of view is that the logical method of interrupting the painful impulses is through the upper four or five dorsal ganglia and not through the cervical ganglia. This has led to operations consisting of alcohol injections of the upper dorsal nerves or extirpation of these nerve roots. The former type of operation carries with it almost no surgical mortality but often results in uncomfortable pains due to alcohol neuritis, whereas the latter is an elaborate surgical procedure which, when carried out on patients with angina, will entail an appreciable immediate operative risk. It appears, however, that alcohol injections properly carried out can produce worthwhile relief for varying lengths of time in about 50 per cent of the cases and that evulsion of the dorsal ganglia can result in 100 per cent relief in those who survive the operation.

As a matter of practice, although these operations are not commonly undertaken, there still may be a proper limited field for their application. I recall a recent instance in which a boy, sixteen years of age, was suffering from intolerable attacks of angina as a part of marked rheumatic aortic valvular disease. The heart was markedly enlarged but there was no congestive heart failure. The boy's condition was pitiful for he had terrific attacks of anginal pain accompanied by palpitation. During these spells the blood pressure would rise markedly. The pain was very severe, would spread across the upper chest, make the boy cry and beg for relief. Nitroglycerin would alleviate and shorten the distress. These attacks occurred frequently even while the patient was at rest in bed. We contemplated surgical treatment as all medical methods had failed. The possible procedures that occurred to us were a simple decompression of the chest as there was marked cardiac hypertrophy with a bulging of the precordium, a thyroidectomy or a complete left-sided cervical sympathectomy. It was decided to try the latter. The therapeutic result was most gratifying. The painful attacks disappeared. Although the boy had spells, they consisted of palpitation and the terrible agony that previously accompanied them was gone. Instead of being a most distressing, bedridden cardiac cripple he was able to return to school, gained weight and was happy. Both the boy and his mother constantly expressed their joy and thankfulness to us for the relief that was obtained and gave us the kind of satisfaction that only too rarely comes to a heart specialist. Similar favorable results in cases of severe angina accompanying aortic valvular disease have been reported following alcohol injections of the dorsal ganglia. I have had other instances in which, after such operations, patients who previously had been greatly handicapped were improved and enabled to walk and carry on without pain. The puzzling feature of all this is that the same operation often proved to be a complete failure in cases that seemed promising. It appears, however, that these operations may



yet be indicated in selected cases for the relief of pain, although there is no evidence that they prolong life or that they prevent the subsequent occurrence of sudden death or coronary thrombosis.

A more recent development in the surgical relief of angina is complete thyroidectomy. Since 1932, when the operation was first performed, several hundred patients have been similarly operated upon in various parts of the world. Enough time has elapsed to form some estimate of the results obtained. The purpose of this procedure was to diminish the work of the heart and to eliminate any nervous or "toxic" effects that the thyroid gland has been suspected of producing. It was well known that, in cases of thyrotoxicosis associated with angina, subtotal thyroidectomy caused marked improvement. The operation was therefore applied to patients with a normal thyroid gland.

Observing the results obtained during the past years, I have come to the following conclusion: In severe angina the operation eliminated entirely or reduced the frequency of attacks in the great majority of cases. The surgical mortality was very small (2 to 5 per cent). There were serious complications following the operation in some instances. Postoperative tetany or aphonia was extremely rare. Occasionally distressing psychoses developed. The resulting mild but protracted myxedematous state led to definite disabilities such as rheumatic pains, feeling of coldness and fatigue, weakness, change in temperament and physical appearance, and other complaints. All these sequelae detracted from the value of the operation. Despite this, many patients were considerably helped and were thankful for the relief of pain. Others lived long enough to experience a return of anginal pain which was difficult or impossible to relieve. Because of the frailties of human nature some of these latter forgot the fact that before the operation their pains were insufferable and that they begged for relief at any cost. The final result is that at present I do not advise total thyroidectomy for angina pectoris. Alcohol injections, on the other hand, when properly and successfully performed, have no disadvantage comparable to the myxedema resulting from thyroidectomy. At present alcohol injections are to be regarded as preferable, in selected cases, if expert surgical technique can be obtained.

Finally, different attempts have been made to actually increase the coronary circulation by surgical measures. Beck sewed the left pectoral muscle to the pericardium overlying the left ventricle. O'Shaughnessy anastomosed a portion of the omentum through the diaphragm to the ventricle. In both instances elaborate experimental work was first carried out in animals which seemed to show that new blood vessels developed in the heart muscle. Later a considerable number of patients with coronary artery disease were subjected to these operations. Finally the left coronary sinus has been ligated by Fauteux in a small group of patients with coronary artery disease. Here also the purpose was to encourage increased arterial supply to the myocardium. It is too



early to conclude whether favorable results have been obtained. For the present the problem must still be regarded in the experimental stage. It is a heartening sign that numerous attempts at surgical treatment for chronic heart disease are being made, for it is the last important organ for the surgeon to explore.

### CORONARY THROMBOSIS

The clinical recognition of acute coronary thrombosis forms one of the most interesting chapters in the history of medicine. Although there were isolated instances in which a clinical antemortem diagnosis of occlusion of a coronary vessel was made, such as that by Hammer in 1878, and although there were other reports that touched upon the question in one way or another, the first publication that discussed the clinical features which might help us in differentiating an attack of coronary thrombosis from one of angina pectoris was by Obrastzow and Straschesko in 1910. A second one by Herrick appeared in 1912. It only then became clear that there were certain findings obtainable antemortem which differentiated the two conditions. In fact Herrick first emphasized that the condition need not be fatal. Before this there was a great deal known concerning the condition from a pathological point of view, but the clinical findings in the living patient remained entirely confused and obscured in terms like "status anginosus" and "myomalacia cordis." It is difficult to understand how the great clinicians of the past, many of whom had had abundant post-mortem experience, could have overlooked this problem, especially when it is appreciated that the recognition of coronary thrombosis developed from a purely clinical study of patients without the use of any of the elaborate procedures or laboratory methods that characterize the modern era of medicine.

Despite these publications in the early part of the second decade of this century there were very few physicians who became familiar with this condition for some years. This important work seems to have been overlooked for a decade or more, except by a very few. In 1916 I had the opportunity of seeing two cases of acute coronary thrombosis, both presenting the picture of an acute surgical abdomen. In the first case I advised immediate exploration and the surgeon agreed to operate. This patient died on the operating table and post-mortem examination to our amazement showed an acute coronary thrombosis and infarction of the myocardium. The second case was seen several months later and recalling the above experience a clinical diagnosis of coronary thrombosis was made. The patient died a short time later and postmortem examination confirmed the clinical diagnosis. These were my initial experiences with coronary thrombosis, a condition which has interested me ever since.

In considering the etiological factors, all that has been discussed under angina pectoris applies more or less with equal force to coronary



thrombosis and infarction of the myocardium. All those influences that are conducive to or associated with arterial disease naturally form the background upon which coronary thrombosis develops. Most important is the familial factor which is so prevalent. Males overwhelmingly predominate. The majority have had a previous hypertension. A large number have had diabetes. Only a few are syphilitic. Some have had conditions like gout, Buerger's disease, polycythemia and hyperthyroidism. An occasional patient who had a rheumatic valvular lesion had in addition disease of the coronary arteries. The great majority of patients had a previous history of angina pectoris either of short, but more often of long, duration. Frequently this early history of angina is overlooked and the sudden attack of coronary thrombosis is then regarded as the first indication of any serious heart disease. I have frequently elicited such definite evidence by direct questioning when it had previously been assumed by both the patient and the physician that all was well. There are, however, many instances in which an attack occurs like a bolt from the blue without warning and in which it can clearly be said that no element of angina preceded it.

Conditions associated with an abrupt fall in blood pressure or marked slowing of the circulation may be conducive to the development of acute myocardial infarction with or without coronary thrombosis. Occasionally coronary thrombosis may develop as a sequence of shock that follows a brisk hemorrhage from a peptic ulcer. It also may occur during or following any surgical operation if there is a significant fall in blood pressure. This complication is particularly to be feared when spinal anesthesia (Fig. 145) is administered and a fall in blood pressure is not prevented or quickly controlled. It is fair to assume that the hazard does not exist except in those patients who already have coronary sclerosis with vulnerable blood vessels. This explains the occurrence of such accidents in elderly patients subjected to prostatectomy and in old debilitated individuals who are in a state of shock for any reason. Attacks of cerebral failure may occur as a result of the same mechanism that has just been discussed.

Coronary thrombosis is related to angina pectoris in much the same way as an occlusion of a vessel of the leg with gangrene is related to intermittent claudication. The anginal state may be regarded as a transitory one leaving the heart in practically the same condition after an attack as before. When a partial or complete occlusion occurs, the muscle supplied by that vessel suffers to a lesser or greater degree. Sometime during the life of those suffering from angina a thrombosis of a coronary artery is apt to occur. This is the common cause of death in angina, although not a necessary one. There are a few cases of angina even among those in which death occurs instantly, in which no acute thrombus can be seen. Such hearts in my experience have always shown significant atheromatous changes in the coronary arteries. In fact I have never seen a case of angina in which the coronary arteries



were normal except when there was one of those associated conditions discussed above, like aortic valvular disease, that adequately explained the condition. However, there have been a few cases of angina reported by others in which the coronary arteries were regarded as normal.

It has always been puzzling to explain the precipitating causes of an acute thrombosis. There is both clinical and pathological evidence to make one believe that it occurs at the site of a previous sclerotic or atheromatous area in a coronary vessel. The occlusion, partial or complete, then occurs rather suddenly. It is not due to an embolus dislodged elsewhere, for coronary emboli are very rare and apt to be associated with vegetation at or near the aortic valve. Do further platelet thrombi develop on the roughened surface of the vessel which leads to greater narrowing of the channel? This seems reasonable but does not quite explain the extreme suddenness of some of the attacks. Or does the final occlusion result from the rupture of a miliary sub-endothelial atheromatous "abscess" as some experimental studies of Leary suggest? The latter hypothesis would explain the abruptness of the symptoms better than the former. Another possible mechanism that has been postulated is that minute hemorrhages first occur within the layers of the wall of the coronary arteries and these are followed by changes in the vessel wall, narrowing and thrombosis. Possibly all mechanisms are involved in different cases.

A new method of studying the coronary arteries by a special type of injection devised by Schlesinger has thrown considerable light on this problem. It has revealed that many thromboses occur that are not recognized in life and that are not accompanied by the clinical features of an acute attack to be described shortly. It has also been found that thromboses occur without infarction and that infarction can also take place without thrombosis. Most hearts have shown two or more occlusions, even as many as ten in rare instances. This technique has revealed many new, interesting and important clinical pathological correlations.

Another perplexing aspect of coronary thrombosis is the frequency with which a particular part of the coronary system is involved. A common site is in the descending branch of the left coronary artery about 1 inch from its origin. Sclerotic changes and thrombosis are so common at this particular spot as to make one suspect that a mechanical factor is involved. Is it possible that as the heart contracts there is a greater bend or torsion of the artery at this point? If this were so it could throw some light on the hereditary factor that is so striking in this disease. We do inherit specific anatomical characteristics such as the color of the iris, configuration of the nose, etc. May not those several members of a family with early coronary artery disease inherit a peculiar architecture of these vessels which puts them under greater mechanical strain as a result of the normal twisting and bending that



goes on with the motion of the heart? This presents an anatomical and physical problem about which we have entirely too few data.

**The Clinical Picture.**—Before taking up the clinical aspects of acute coronary thrombosis it will be helpful to review briefly the pathological process that is taking place. When a coronary vessel becomes partially or completely occluded the area of heart muscle supplied by that vessel becomes infarcted. Early in the process there is extravasation of blood, then muscle fiber necrosis and finally repair by scar tissue. If the involved area extends to the periphery a localized aseptic pericarditis may develop, and if it extends inwardly the endocardium may be involved, resulting in a large secondary ventricular mural thrombus attached to the area of infarction. The local area of necrosis may become sufficiently softened to perforate during the early days or the wall may be weakened to such an extent that a localized aneurysm of the ventricle results. When satisfactory healing takes place due to the anastomosis of collateral vessels, which is much more abundant in the heart than was formerly thought, a healthy scar forms. This simple review enables one to picture some of the clinical events that occur.

Attacks of coronary thrombosis seem to occur more frequently at rest than during effort. In this respect it differs from angina pectoris. It is a common experience to find that the patient is aroused from sleep with the attack. On close questioning many will confess that during the preceding day or two they had not felt quite so well and may have had more or less milder discomfort in the chest. Occasionally one may suspect that a coronary thrombosis is impending when a patient who previously had ordinary attacks of angina promptly relieved by rest or nitroglycerin suddenly starts having attacks without effort, lasting a half hour or so and not responding to nitroglycerin; or if similar severe symptoms occur without preceding angina, an outspoken coronary attack not infrequently follows in a few days.

In its typical form the pain of coronary thrombosis becomes very severe, almost unbearable. The location and severity of the pain varies a great deal in different cases just as there is a striking variability of most of the clinical features of the disease. Although no single sign or symptom is found in all cases, there generally is enough evidence of one form or another to make a fairly definite diagnosis. The amount of pain may vary from none at all to the most severe agony any mortal can suffer. It takes the form of a pressure or terrible crushing or squeezing sensation. Its location most often is in the middle of the chest centering around the sternum, or between the two nipples. It can either begin in or even be limited to the upper epigastrium near the ensiform. It often radiates to the upper midback, shoulders and arms. It must be borne in mind that in some the distress is not very marked and in fact there are a few who are not prevented from carrying on their activities throughout an attack. The duration of the pain is also variable, lasting from an hour or two to several days. Generally after six or



twelve hours the severity has abated although a milder soreness persists. Not uncommonly after the pain has subsided it returns in varying degrees for several days or longer.

Breathlessness may be the presenting complaint, particularly in those occasional instances in which pain is very slight or absent. Although dyspnea is not present in most cases there are a few in which the clinical picture is one of acute pulmonary edema. Even when the respiratory symptoms are not so fulminating, orthopnea or other evidence of embarrassed breathing may be apparent.

In the great majority of instances there is a peculiar and rather characteristic appearance of the patient during the height of the attack. He seems to be in shock. The skin is cold, moist and takes on a gray ashen color. The face is anxious and one is quickly aware that something profound has happened. In the early hours and days sweating is often very marked, resulting in dehydration and a loss of salt from the body. In fact, a very moist skin may persist for weeks and gives one the impression that the underlying process of myocardial infarction is still active or insecure. In some atypical cases a moist skin may be the first clue that the illness which was painless and consisted mainly of weakness is due to an acute coronary thrombosis. Great weakness is often very striking even when there are but few other complaints. With this there is generally a fall in the blood pressure. This fall occurs in varying relation to the onset of the attack. Sometimes the first reading that the physician obtains is extremely low or the patient may already be pulseless. Not infrequently the blood pressure is still elevated during the first few hours and falls subsequently. On very rare occasions no significant change takes place at all. In the typical case the systolic pressure that was around 170 mm., gradually falls to 110 mm., although this change may take place over several days. With the progress of the case it may return to its previous level or be permanently lower to a greater or lesser extent. Practically always the pressure returns approximately to the original level during the following year or two.

Gastro-intestinal symptoms are often prominent and may be troublesome from a diagnostic or from a therapeutic point of view. The abdominal pain and tenderness which occur in a few cases resemble an acute surgical condition. Even slight icterus may be present. Nausea, vomiting and distention may be distressing and at times resemble the symptoms of acute intestinal obstruction. Vomiting is very common during the first hours of the attack and may become even more prominent after the frequent injections of morphine are given which are necessary to control the pain. Occasionally annoying hiccoughs develop. Usually all these symptoms disappear after one to several days.

A slight fever and leukocytosis are very common accompaniments of coronary thrombosis. They both require some time to develop and are generally present in twenty-four to forty-eight hours, although they may be found at times as soon as six hours after the onset. The fever



is often overlooked if the temperature is taken by mouth as these patients are in shock and the periphery of the body may be actually cold in the presence of a true fever. This error will be avoided if a rectal temperature is taken. I have often found the latter to be  $101^{\circ}\text{F.} \pm$  when the mouth temperature was only  $98^{\circ}\text{F.}$  The slight fever lasts from three to seven days, rarely longer. The leukocytosis ranges around 15,000 to 20,000 and also lasts several days. The sedimentation rate of the erythrocytes is also increased, probably as a result of the myocardial infarction. This increase is apt to occur a few days after the onset of the attack and may persist for two weeks or more even after the fever and leukocytosis have disappeared. Occasionally the sedimentation rate may indirectly aid in differentiating an attack of coronary thrombosis from one of angina pectoris or other conditions or may help in the decision as to whether an extension or aggravation of the myocardial infarction has taken place.

The findings on examination of the heart are variable. The sounds generally diminish strikingly in intensity so that at times they are almost inaudible. The rate becomes accelerated to about 100 to 120 and in many instances a definite gallop rhythm can be heard. Just as is true of all these individual features, there are numerous exceptions. Occasionally there is no acceleration of the heart rate. A small number of patients will show a transient pericardial friction rub. When this occurs it is extremely helpful in diagnosis but its development will depend upon the location and extent of the myocardial infarction. Almost any of the disturbances in rhythm with which we are familiar may develop during the early days following an acute coronary thrombosis. When they do occur most of them are transient and the normal rhythm returns. Of the important arrhythmias, transient auricular fibrillation is most common. Auricular flutter is rare but ventricular tachycardia is not uncommon. Extra systoles are also common. Some patients will show partial and a few will develop complete heart block even with Adams-Stokes' attacks of syncope. Conduction defects are more apt to develop with posterior lesions. The apex impulse disappears as a result of the feeble contraction. Although one might think that with such a profound injury to the cardiac mechanism dilatation would occur, *x-ray* studies have shown no such change.

The examination of the lungs will frequently show some rales at the bases, especially on the right side. Occasionally typical evidence of acute pulmonary edema will be found with moist or bubbling rales all over the chest. In fact, an attack of coronary thrombosis may be ushered in by this type of onset even without chest pain. Sudden dyspnea may, therefore, replace sudden pain as the presenting complaint. Cheyne-Stokes breathing is very common even when there is no subjective respiratory distress. The abdominal examination is generally not remarkable, although in a few cases the findings resemble an acute surgical condition for there may be tenderness and muscular spasm



in the epigastrium or right upper quadrant. In some cases at least these findings are due to an acute passive congestion and enlargement of the liver with the accompanying tension on the liver capsule. In one case in which the abdomen was explored as a result of a mistaken diagnosis this was observed.

The urinary findings deserve some mention. Many patients will show marked oliguria and some will void practically no urine for twelve to twenty-four hours. This is the result of the low blood pressure and the state of shock. The urine that is voided is concentrated and may show an appreciable amount of albumin and numerous cells and casts. Glycosuria is very common, first because so many diabetics develop coronary thrombosis and because others who never had glycosuria before may temporarily show sugar in the urine as part of the coronary attack. Some of the latter are possibly mild or potential diabetics but it seems that in others, who temporarily show glycosuria, it is entirely precipitated by the heart attack. What is important to bear in mind is that the urinary findings indicative of renal disease need give us no great concern as they always clear up if the circulation improves. The same may be said of the glycosuria. It rarely if ever requires insulin during the acute stages. In general it is better to disregard the diabetes unless, as very rarely occurs, there is a significant acidosis as shown by a lowering of the carbon dioxide combining power of the blood.

Electrocardiography has been a most important aid in the diagnosis of coronary thrombosis. Although Herrick very early in his work suspected that certain electrocardiographic changes might possibly be the result of coronary thrombosis, it was Pardee who first established the fact that during the early hours or days after an attack, the ventricular complex may take on a peculiar form. This consisted of a high take-off of the T wave from the Q-R-S complex before the latter reaches the iso-electric line in one or another of the customary three leads. This does away with the normal short flat interval between the initial and final phases of the ventricular complex. It has also been found that rapid alterations in the complexes occur during the succeeding days or weeks following an attack. The T waves become rounded and dipped (cove-shaped) and finally peculiarly inverted (Chapter 21). At any one particular time these tracings may appear essentially normal but if a series of several electrocardiograms is taken during the first two weeks, most if not all will show definite or at least suspicious alterations. The introduction of the fourth or chest lead by Wilson, Wolferth and Wood has further increased the usefulness of electrocardiography, for there are some instances in which the area of infarction is so located that the customary limb leads fail to show any significant aberrations while the chest lead will bring forth definite evidence of heart muscle injury.

Electrocardiography has not only been valuable in diagnosis, but



has enabled us to predict in many instances the exact location of the area of infarction. Injuries to the anterior part of the heart generally occurring in the lower lateral portion of the left ventricle near the apex and resulting from a thrombosis of the descending branch of the left coronary artery are associated with one type of electrocardiogram. Similar injuries to the posterior part of the ventricle, either resulting from a thrombosis of the circumflex branch of the left coronary artery or of the right coronary artery, produce a different set of changes. In the former the high take-off of the T wave with subsequent inversion of the T wave and the appearance of a Q wave occur in Lead I. In the latter type these similar changes occur in Lead III. Wilson has indeed shown that while the changes in the T wave are often transient with even complete restoration of the normal appearing upright T wave, the changes in the initial ventricular complexes, notably the Q wave, are apt to be permanent. In this way one may be enabled to suspect months or years after an attack, that one previously had taken place. In this regard the information obtained from the fourth lead promises to be even more valuable. It has been found that normal individuals practically invariably have a prominent R wave in Lead IV and what seems to be of less importance an upright T wave. It now is believed that the absence of any R wave whatever in Lead IV, especially if associated with an inverted T wave, denotes a previous infarction of the anterior part of the ventricle. The finding of normal curves in Lead IV does not eliminate the diagnosis of coronary thrombosis, because involvement of the posterior part of the ventricles produces no such characteristic changes. Apart from these electrocardiographic changes there are others that are met with in coronary thrombosis that are by no means distinctive of the condition. Further electrocardiographic details will be taken up in Chapter 21. All forms of conduction disturbance are found, such as partial or complete heart block and bundle branch block. Also in some the ventricular complexes are of unusually low amplitude. These findings may be of importance in some cases. Unless the changes are quite characteristic, caution must be exercised in accepting alterations in the complexes as unequivocal evidence of myocardial infarction, as they may occur in a variety of other conditions such as pulmonary embolism, acute dissecting aneurysm, acute nephritis, uremia and rheumatic carditis. Although some of these studies are of quite recent origin and require final definition and confirmation, I believe much may be expected from the electrocardiographic diagnosis of coronary artery disease.

In an uncomplicated case of coronary thrombosis of moderate severity the patient is apt to become free from pain after the first day and thereafter to remain quite comfortable. In other patients pain returns irregularly over a much longer time. Those whose respiratory function becomes embarrassed may have marked dyspnea and orthopnea. The various irregularities of the heart previously mentioned may suddenly



change the clinical status and require special attention. At any time during this illness sudden death can occur either as a result of ventricular fibrillation or rupture of the softened infarcted area of the ventricle or possibly from complete heart block. Ruptures do not usually occur until about the fourth to seventh day. Another type of complication is embolism. In many of these cases a fresh mural thrombus develops in one of the ventricles, generally the left, contiguous to the area of infarction. Portions of this thrombus may become dislodged and occlude other vessels producing secondary embolic lesions. If they come from the left ventricle, hemiplegia, renal, splenic or mesenteric infarcts may result or gangrene of one of the extremities. If they arise in the right ventricle, pulmonary embolism or infarction occurs. These are most apt to take place after the first few days. The pericarditis that occasionally is found requires no particular concern as it almost never results in empyema or pericardial effusion. It is evident, therefore, that the exact course which any individual case may take can vary greatly.

Fatalities may take place with great suddenness even when everything seems to be going most favorably. In other patients there is a gradual weakening of the pulse with a low blood pressure, marked weakness and a quiet, peaceful end. In some there is a great deal of dyspnea and air hunger.

When recovery takes place we also have different sequelae to be anticipated. There are some who, having suffered from angina pectoris, either have no symptoms at all after an attack of coronary thrombosis or have much less trouble than they had prior to the attack. These patients are apt to be the ones who previously had hypertension in whom the pressure becomes permanently lowered after an occlusion of one of the coronary arteries. In others anginal attacks return with the same frequency and in some angina pectoris may appear for the first time after a coronary attack. There is a large group which, never having shown any dyspnea or congestive heart failure, first begins to manifest these symptoms after an attack of coronary thrombosis. Occasionally this occurs during the first week following the attack but more commonly the patient becomes ambulatory and only months or years later begins to have general circulatory failure.

A localized aneurysm of the left ventricle with thinning and weakening of the wall occasionally results from a previous infarction. Curiously enough, rupture of the ventricle, which is by no means rare during the early days of an acute attack, rarely if ever occurs at the site of such chronic fibrosed ventricular aneurysms. They are present after recovery has taken place and the patient is ambulatory, and the condition is compatible with a fairly satisfactory state of the circulation. The diagnosis of ventricular aneurysm will rest on a previous history of myocardial infarction, the finding of a visible and palpable apex impulse well inside the outer border of dulness, a diminished first heart sound,



and x-ray examination. The latter will show a localized bulge and on fluoroscopic or kymographic examination this bulge will be observed to expand outwards with systole as the neighboring musculature contracts inwards.

**Prognosis.**—The prognosis for patients with acute coronary thrombosis with myocardial infarction is variable. When large series of these patients were first studied it seemed that about 50 per cent recovered and the other 50 per cent died during the acute attack. The true figures are now much more favorable because in the former studies only classical, easily recognized cases were included. Now, with more advanced methods of diagnosis, many milder cases can be included and the immediate mortality will vary between 15 and 25 per cent, depending on the accuracy of the diagnosis.

In an extensive review of the immediate mortality certain points of interest came to light. Anterior and posterior lesions were equally serious. The mortality was lower if significant electrocardiographic changes were slight or absent, was slightly higher for women than men and was definitely higher in older than younger individuals. A previous history of angina improved the immediate prognosis, while a pre-existing hypertension made it worse. A marked fall in blood pressure, especially if the level was maintained below 80 mm. for some time, made the outlook quite grave. The severity of dyspnea was more ominous than the degree of pain. In general, the immediate mortality was greater if the customary clinical features of acute coronary thrombosis, *i.e.*, a higher fever, more rapid pulse, greater leukocytosis, more profound shock, etc., were more prominent.

After recovery from an acute coronary thrombosis had taken place the duration of life was found to vary greatly, the average being about three and one-half years. There was little difference in the length of survival between subjects with anterior and those with posterior infarctions. Those with only minor alterations in the electrocardiograms lived longer. One fourth died in one year, one-half in two years, three-fourths in five years and the remainder at varying intervals up to more than twenty-five years. Two-thirds had angina some time after the attack of coronary thrombosis and about one-fourth developed congestive failure. The latter complication occurred more frequently with anterior than with posterior infarction. About 30 per cent resumed essentially full duties for varying lengths of time and the remainder were more or less restricted. The survival period for women was much shorter than for men and it was much longer for younger subjects than for older ones.

The knowledge that some can recover and carry on in good health for a long time, even fifteen years or more, permits us to encourage our patients. Furthermore, satisfactory recovery from a second and even multiple attacks occurs but with increasing rarity. In general the prognosis should be guarded but always hopeful for even in the face of an



extremely severe and desperate attack satisfactory recovery may take place.

**Differential Diagnosis.**—There are several conditions that may at times become easily confused with coronary thrombosis. Of first importance are the conditions that resemble acute surgical states of the abdomen, such as gallstone colic, perforated peptic ulcer, acute pancreatitis, acute appendicitis and acute intestinal obstruction. All the diagnostic methods available may be required to arrive at the correct diagnosis in such cases and despite all the care there will be rare instances in which errors will be made. The presence of dyspnea with the attack and the radiation of the pain to the sternum or to the arms may be helpful in differentiation. The electrocardiograms may be the turning point on which a diagnosis will rest. There is also the opposite danger of overlooking an acute surgical condition requiring immediate abdominal operation in our enthusiasm to detect instances of coronary thrombosis. For it must be borne in mind that patients with known organic heart disease, as well as those who are only suspected of having it, may also have these very surgical conditions that require an operation. Under certain circumstances, when the diagnosis is in doubt, it probably would be safer to explore the abdomen even at the risk of an unnecessary operation rather than to overlook a perforation of the stomach.

Another condition that may closely simulate acute coronary thrombosis is pulmonary embolism or pulmonary infarction (acute cor pulmonale). In both there may be sudden circulatory collapse, dyspnea, a rapid thready pulse and cyanosis. Pain in the chest is less common with pulmonary embolism, although it does occur occasionally and is particularly disconcerting when it takes place on the left side. Contrariwise, in both conditions pain may be absent. During the early hours after a pulmonary embolism, there is no hemoptysis and examination of the lungs is apt to be of no help in diagnosis. Both conditions may occur after surgical operations, but it will help to simplify matters if a cause for pulmonary embolism such as phlebitis can be detected. Distention of the veins of the neck, although not an absolutely distinguishing physical finding, is more likely to be present with a pulmonary than with a coronary attack. In pulmonary embolism the pulmonary second sound is often accentuated and there is a systolic murmur in this area probably due to the dilated pulmonary artery and the increased pulmonary pressure proximal to the embolus. Finally the electrocardiograms may prove valuable in diagnosis. Minor changes in the ventricular complexes are common to both, but outspoken alterations like a very high take-off of the T wave or a sharp rounded inversion of the T wave occur only with cardiac infarction. Occasionally the electrocardiograms are sufficiently distinctive to make a fairly definite diagnosis of acute pulmonary embolism (Chapter 21, Figs. 125, 126, 127).



In reviewing the differential diagnosis I need but recall errors that have occurred, some of which I have made myself. In one instance some years ago I made the diagnosis of coronary thrombosis because of sudden suffocation and collapse occurring in a man sixty years of age. The patient seemed to be in shock with a rapid thready pulse. Although there was very little pain I thought he had had a coronary accident. It was not until the next day that I realized that one side of the chest was not expanding with respiration and there were practically no breath sounds on that side. He had a complete pneumothorax from which he recovered very satisfactorily.

Another more common difficulty in diagnosis is the differentiation of pneumonia and coronary thrombosis. The fairly acute onset of chest pain, cough, dyspnea, cyanosis, the development of fever, leukocytosis, rapid pulse and rales in the lungs occur in both conditions. There are no significant alterations in the electrocardiograms in pneumonia, however. This differential diagnosis at times is very difficult and I recall erring in both directions, considering a case one of pneumonia when it was coronary thrombosis and in another making the opposite mistake. Electrocardiograms may serve to differentiate the two. No doubt many elderly patients considered as having pneumonia really have suffered an attack of coronary thrombosis.

Diabetic acidosis and coma may be simulated by acute coronary thrombosis. Inasmuch as the latter often occurs in diabetic patients glycosuria is very common. There may also be an associated acidosis with diacetic acid in the urine and a lowered carbon dioxide combining power of the blood. When such findings accompany a state of semi-stupor or complete unconsciousness one is strongly tempted to regard the condition as diabetic coma. All this, however, can result from an attack of coronary thrombosis. Inasmuch as insulin may be harmful to patients with coronary artery disease great care must be exercised in differentiating the two conditions. If the blood sugar is not high, insulin should not be given and even when found to be elevated only small doses should be used unless it is certain that diabetic coma is present. In general I have very rarely found it necessary to use insulin in such patients.

Hamman has called attention to a condition that may closely simulate acute coronary thrombosis. He called it "spontaneous interstitial emphysema of the lungs." Many of us must have confused these two conditions in the past. Such patients are suddenly stricken with violent pain over the precordium with radiation to the left shoulder and left arm and there may be an acceleration of the pulse, a leukocytosis and slight fever. All this results from the rupture of an air sac in the lung that dissects its way along the bronchi and blood vessels and infiltrates the mediastinal tissues. The air may extend in the subcutaneous tissue to the neck or may reach the pleural space, producing a pneumothorax. The most important and characteristic finding is the presence of



unusual clicking, grunting or crunching sounds synchronous with the heart beat. These sounds may be influenced by respiration and may be entirely absent with the patient flat on his back, only to be brought out in the left lateral position. In some, the x-ray will show air in the left pleural cavity or in the anterior mediastinal spaces. This condition is essentially benign, as complete recovery is apt to occur. The only treatment necessary is sedation for the pain and a brief period of rest. Its importance lies in the fact that it needs to be differentiated from coronary thrombosis and it impels us to watch carefully for these unusual auscultatory findings. It also must not be confused with noises that are rarely heard in cases of diaphragmatic hernia. Such splashing "heart noises" result from the beating of the heart against the stomach or intestines, which may lie in the chest.

There is a group of patients in which the early symptoms of the acute stage are so mild that they are disregarded and the subjects present themselves as medical problems only after an embolus has occurred, particularly when a hemiplegia results. Some cases that are diagnosed cerebral hemorrhage fall into this group. The possibility that a hemiplegia may be due to an embolus dislodged from a left ventricular mural thrombus following a coronary thrombosis should always be considered when it occurs in a patient who has a low blood pressure. Even in elderly patients cerebral hemorrhages of the ordinary type, excluding those resulting from aneurysms of the cerebral vessels, rarely if ever occur without hypertension. A consideration of all the diagnostic points may be necessary in order to arrive at the correct diagnosis.

There are atypical cases of coronary thrombosis in which extensive experience will be necessary to avoid making erroneous diagnoses. In particular is this true of the occasional instances in which pain does not occur. Here there is apt to be sudden breathlessness and a feeling of exhaustion. In such cases the dyspnea will be found to be out of proportion to other evidences of heart failure. When this is the case, a low blood pressure, if it was known to be high before, together with other features, such as changes in the electrocardiograms, will give one the proper clue.

When the coronary vessels are slowly narrowing, infarctions of the heart may occur without any acute episode. This may take place in patients who have progressive heart failure and are regarded as suffering from chronic myocarditis. Even here a proper survey of all the data, especially obtaining a history of early angina pectoris and the study of the electrocardiograms, may enable us to anticipate the true pathological state.

Considerable interest has developed in the diagnosis of dissecting aortic aneurysm which is often confused with acute coronary thrombosis. The pain in the former comes with even greater suddenness than in the latter. The pain in the chest is crushing, may extend through to the back and at times even to the legs. Hypertension is almost invariably



found and it tends to persist after the attack. Fever and leukocytosis develop but there are none of the irregularities of the heart or a friction rub that occur in coronary thrombosis. Although the electrocardiograms in most cases remain unchanged and fail to show significant alterations in the ventricular complexes, T wave changes resembling myocardial infarction may appear as a result of pressure on one of the coronary arteries by the aneurysm. In fact, some of the peculiar findings in this condition will depend on the location of the dissecting aneurysm and the direction which the dissection takes. After splitting the wall of the aorta, the aneurysm and blood clot may extend down the abdominal aorta even to the iliac vessels. In its course it may compress any of the arteries that are given off from the aorta and produce a variety of symptoms. Giddiness and blurring of vision may result from the effects on the carotid arteries, anuria or hematuria from involvement of the renal vessels and pain and numbness in the legs from occlusion of the common iliac artery. Paralysis of the limbs may develop from involvement of the spinal cord. The peripheral pulse in one or another of the limbs of the body may disappear. Sudden death frequently occurs from rupture of the aneurysm. Although the two conditions have many clinical features in common, careful consideration of the finer differential points involved, especially the direct findings in the heart, will generally suffice to distinguish coronary thrombosis from dissecting aneurysm.

Incomplete tear or rupture of the aorta also occurs that may or may not be followed by dissection. It is extremely difficult to recognize this condition clinically. It may account for attacks of choking or suffocation or mid-chest pain occurring in patients with hypertension. These tears are generally horizontal and when they are present quite close to a commissure of the aortic valve, it appears that the valve may sag and result in aortic insufficiency. This is thought to be the explanation of an aortic systolic and diastolic murmur that may develop in some patients with incomplete rupture of the aorta. Healing of these tears can take place or dissection may develop hours, days or, possibly, even months later.

With the great increase in knowledge concerning coronary thrombosis that has taken place in recent years, the diagnosis is now being made too frequently. Whenever the evidence is not entirely convincing one should search for other conditions. Apart from gallbladder disease and gastro-intestinal disturbances already mentioned, bleeding peptic ulcer and diaphragmatic hernia must be considered. The shock, weakness, rapid pulse, and fall in blood pressure resulting from hemorrhage can resemble a painless coronary thrombosis. Similarly the peculiar distress in the chest that may occur with a diaphragmatic hernia, especially in women, may simulate a coronary attack. In neither case, however, will there be found reliable evidence of myocardial infarction. Herpes zoster, arthritis of the spine, carcinoma of the lung and syphilitic aneurysm of the aorta are other conditions not to be



confused with coronary thrombosis. The list of diseases that may enter into a differential diagnosis is almost endless, as is well illustrated by a recent experience in which coronary thrombosis was confused with the very rare condition called diaphragmatic flutter.

Finally the differentiation of simple angina pectoris and coronary thrombosis needs consideration. Generally this is not difficult. All the features discussed in the preceding pages under these two headings help to distinguish the one from the other. Although most attacks of angina last only a few minutes some continue for fifteen minutes or more. It is not always possible to detect evidence of coronary thrombosis when attacks last longer, although some of these no doubt result in myocardial infarction. In many cases, on postmortem examination, there have been noted several isolated areas of fibrosis from old infarction, in which a corresponding number of attacks cannot be distinguished from a clinical point of view. Furthermore, I see patients with typical angina pectoris in whom I can obtain no evidence whatever of an attack resembling typical coronary thrombosis who yet show definite evidence in the electrocardiograms of a previous myocardial infarction. The term "acute coronary insufficiency" may well be applied to attacks of prolonged coronary pain in which no evidence of myocardial infarction appears. The most careful observation of patients with so-called "attacks" of severe angina pectoris, watching for a slight fever or for significant alteration in the electrocardiograms, will be necessary to arrive at the correct diagnosis.

**Treatment.**—There is hardly any other condition in the general field of heart disease in which it is more difficult to appraise the value of specific measures of therapy than in the treatment of acute coronary thrombosis. Events occur with such suddenness that one may be too ready to attribute as a cause of the result, whether favorable or unfavorable, the last procedure employed. Notwithstanding this there are certain methods of treatment that for the present meet with general acceptance and others that may be regarded as still in the experimental stage or at least as open to doubt.

Of first importance is the relief of pain. This is best obtained by the liberal use of morphine. The amount necessary will vary from one dose of 0.015 gram ( $\frac{1}{4}$  grain) subcutaneously to many such doses. When the pain is very severe and persisting morphine should be repeated in one-half hour or so and at times a grain or more will be necessary. Morphine should be given subcutaneously or intravenously as oral administration will be entirely inadequate. Quite recently papaverine (0.05 to 0.1 gram given intravenously) has been suggested for severe coronary pain. During the early minutes after the onset, if the patient's condition seems very critical, he should not be moved or even undressed unless it is extremely inconvenient to treat him as he is. These changes are often better made a few hours later. When, as occasionally happens, the patient quickly becomes unconscious and



pulseless the hypodermic use of adrenalin (0.5 to 1 c.c. of 1 : 1000) is indicated and may help to restore consciousness.

Complete mental and physical rest is paramount. The patient should be spared all possible physical movements. Frequent tiring examinations are undesirable. After the initial pain has subsided, sleep should be assured the first few nights, even using morphine if necessary. It is just as well to avoid the use of enemas for one to several days despite the absence of bowel movements. There is very little food consumed during these days and it is not necessary that the bowels should move every day. An enema on the third or fourth day will often be better tolerated. There is very little that can be done for the nausea and vomiting that occur during the first day or so. They are partly the result of the attack and partly brought on by the morphine that is given; generally they subside after the first day especially if narcotics become unnecessary.

Considerable dehydration develops in some of these patients. They often lose a great deal of water and salt with the marked perspiration that occurs and from the vomiting and inability to retain fluids and nourishment taken by mouth. It sometimes is imperative to administer 1000 c.c. of normal salt solution subcutaneously. Some observers have been very favorably impressed by the results of intravenous injection of 100 c.c. of 50 per cent glucose solution. I have not witnessed any striking improvement following this procedure. Another reason for encouraging the intake of fluid is the oliguria that may amount to a complete anuria so common after an attack. There is also some ground to believe that the administration of sodium chloride would be desirable, for the state of shock can certainly be aggravated by the great loss of salt that takes place from excessive perspiration in some cases.

Oxygen may be of use. When there is marked dyspnea and pulmonary congestion the inhalation of oxygen can improve breathing and cyanosis. In fact I have also seen an instance in which severe and persistent pain seemed to disappear promptly after placing the patient in an oxygen tent. During the first hours stimulants like caffeine and adrenalin may be beneficial, the former for respiratory distress and the latter for shock and a low blood pressure. It is difficult to be convinced of their ultimate favorable effects but they seem to be helpful. What is much needed is some method of combatting shock and the low blood pressure that is often present. There is every reason to believe that plasma or albumin administered intravenously might be helpful for the profound state of shock. Although I have used this method, my experience is too limited to draw any final conclusions. From a physiological point of view it is conceivable that an increase in blood volume produced by plasma would be desirable at one time for shock and a decrease produced by bleeding for pulmonary edema at another time, even in the same patient. In a recent case in which the pulse could not be felt, 250 c.c. of plasma given intravenously and



repeated in four hours caused a dramatic improvement and seemed to save the patient's life. Possibly some of the newer preparations, such as paredrine (10 to 20 milligrams, intramuscularly) which has recently been recommended, may prove useful. In this connection it is of interest that paredrine has been shown to produce venous constriction. If this is true it ought to be helpful in increasing venous return, which is much to be desired in shock.

The remainder of the treatment is expectant and directed at complications. Of these, two are rather important from a therapeutic point of view. First is the development of complete heart block with or without Adams-Stokes syncope. This is an infrequent complication but may be controlled very satisfactorily by the hypodermic injection of 0.5 to 1 c.c. of 1 : 1000 adrenalin solution. In some cases it will be necessary to give such injections frequently. In several instances I have given 0.3 to 0.5 c.c. of adrenalin every two hours for forty-eight hours to patients who had this condition and thereby prevented the pauses of the heart that were otherwise occurring, finally observing that the tendency to syncope had disappeared. It has seemed to me that in rare cases such treatment has been life saving.

The other complication to be considered is paroxysmal ventricular tachycardia. This occurs in about 3 per cent of the cases. When it develops it can quickly produce a state of collapse. The heart rate becomes very rapid (180 to 200) and the blood pressure falls still further. The condition can often be recognized by three features. The rapid rate though apparently regular is apt to show slight irregularities which the ear can detect. The first heart sound may change in quality and intensity in different heart cycles and carotid sinus or ocular pressure never produces any slowing of the heart. These points can distinguish ventricular tachycardia from other forms of paroxysmal rapid heart action. Quinidine sulfate and magnesium sulfate are the drugs of choice in controlling this irregularity. Digitalis will not only fail to slow it but will tend to accelerate or to perpetuate it. Quinidine can stop it but the dose required varies considerably. I have seen such an attack promptly stop after one dose of 0.3 gram (5 grains) taken by mouth. In another case it required 1.5 grams (22 grains) to stop an attack and such a dose five times a day for several days to prevent the return of the tachycardia. In two instances it was found that even very large doses of quinidine merely slowed the ventricular rate and failed to abolish the abnormal mechanism. The rate would gradually return to the previously high level as the effect of the drug wore off. In these two cases the hypodermic injection of 0.002 gram ( $\frac{1}{30}$  grain) of atropine sulfate, one hour after a large dose of quinidine had been given orally (while the temporary slowing had taken place), promptly eliminated the ventricular tachycardia. Quinidine may be also given intramuscularly or intravenously. It would appear that the parenteral method, especially the intravenous, would carry more risk. Quinidine sulfate merely



restores the normal rhythm of the heart and controls this complication. It does not prevent the other complications to which the same patient is still subject. Similarly 2.0 to 4.0 grams of magnesium sulfate, given intravenously, may stop these attacks.

There is a final use of quinidine that bears mention. Evidence has been advanced of both a clinical and experimental nature that quinidine sulfate tends to inhibit the development of ventricular fibrillation. It is more than likely that some of the instances of sudden death in coronary thrombosis are due to ventricular fibrillation. I formerly advised giving 3 grains of quinidine three times a day during the first two or three weeks following an acute attack, except when there is any contraindication to its use. The main contraindication is any evidence of defects in conduction, like bundle branch block, or partial or complete auriculo-ventricular block. Inasmuch as quinidine can further impair the conduction apparatus it should be avoided under such circumstances. It is difficult to prove that the routine use of quinidine as has been suggested has any practical merit or that the dose is adequate to produce the effect for which it is given. Only much more extensive experience can answer these questions. At present, however, I give quinidine routinely whenever the patient can be observed intelligently and carefully.

It is the custom of some physicians to give preparations like aminophyllin routinely during attacks. Although there is some experimental evidence to show that such a drug increases coronary flow and that it diminishes the extent of myocardial infarction after coronary arteries are ligated, it is difficult to be certain that it is useful in clinical cases of acute thrombosis. This subject, however, deserves further study. Such a drug has been used even intravenously during the height of pain. I have had some experience with this but it both requires and deserves further controlled observation in clinical cases to establish the usefulness of such medication.

In addition to some of the things the physician is called upon to do there are certain procedures he should not do. When a syphilitic patient has an acute coronary thrombosis it is inadvisable to give any intravenous antiluetic treatment. In fact it is better to disregard the syphilitic aspect of the problem entirely during the first two weeks or so and then to give merely potassium iodide and mercury or bismuth. In these cases it is not at all certain that syphilis is directly related to the coronary thrombosis for it is more likely that the latter is due to the same causes that are at work in nonsyphilitics. Reference has been made previously to the use of insulin in cases of coronary thrombosis. This should be avoided as much as possible because for an hour or two after its use the work of the heart is increased about 20 per cent if the blood sugar drops too low and this added strain may be very serious. It is well known that attacks of angina can be precipitated by insulin and I have seen a rupture of the ventricular wall occur about one-half hour after 10 units were given. Insulin may be given cautiously if the blood sugar



is high and marked fall of the blood sugar can be avoided. Furthermore, if a patient has an acute coronary thrombosis and some other surgical condition, such as an obstructing prostate or an infected kidney that needs removal, it is advisable to delay operation for at least a month or two until a satisfactory recovery of the heart has taken place. There is a final precaution in the care of patients with coronary artery disease. It has been observed that an attack of coronary thrombosis can occur directly after the intravenous injection of the iodine dye that is used for cholecystograms. I have seen two patients with angina pectoris in whom an attack of coronary thrombosis was brought on a few minutes after the intravenous injection of the dye. It is wiser, therefore, when a patient is either known to have or suspected of having coronary artery disease and it is desired to study the condition of the gallbladder, that *x-ray* examination should be made only after oral administration of the dye.

Finally, the question arises whether or not digitalis should be employed in acute coronary thrombosis. There are many theoretical reasons one could give for or against its usefulness. I have always felt that it is more likely to do harm than good. If persistent auricular fibrillation is present it should be given but this condition is very rare, as when it occurs during an attack it is almost always transient. Furthermore, when there is peripheral pitting edema, engorged liver or hydrothorax, digitalis should be administered as in any case of congestive heart failure. This is very rare, however, except after the first few weeks. It is uncertain whether or not it is beneficial in those cases showing rales in the lungs and dyspnea. The drug should be helpful for left ventricular failure but might be harmful for the peripheral failure or shock, and both conditions exist simultaneously in the early days of acute coronary thrombosis. The lack of cardiac dilatation would make one hesitate in using the drug. Although much more statistical evidence will be required before the question can be finally settled, at present it seems best not to use digitalis routinely during the first two weeks of acute coronary thrombosis.

The general nursing care of the patient is very important. Everything should be done to make him comfortable. The diet during the early days should be confined to liquids, gradually returning to more ordinary food in small amounts. Many of these patients are overweight and to obtain a loss of weight during this illness is desirable. It has been advised that a low caloric diet (500 to 800 calories a day) should be used in acute coronary thrombosis and in fact is being advocated in the treatment of any severe or stubborn case of congestive heart failure (Proger and Master). It has been found that this semistarvation diet diminishes the work of the heart and produces other favorable effects on the circulatory dynamics. Patients should be kept at rest for four to eight weeks. Occasionally the period of bed rest or restricted activities needs to be longer. From a purely clinical point of view the four to



eight-week period has been found to be adequate and it is of interest that in large animals it has been observed that two to five weeks are required to establish adequate collateral circulation after experimental ligation of a major coronary artery. It is a general biologic principle that the main stimulus for the development of collateral arterial circulation is a local need for it. In as much as the present evidence favors the view that there are anastomoses from one main coronary artery to another normally, even in infancy, it is not difficult to conceive of the mechanism of recovery from myocardial infarction. Economic and social factors often will determine the exact length of time of convalescence. Whether nurses will have to be employed is also often a matter of the financial status of the patient. It generally is advisable that the patient should not be permitted to go to the bathroom for either bowel movement or urination during the early days. Occasionally it proves to be less of a hardship to permit the use of a commode than that of a bed pan. These details need to be worked out by the physician in individual cases. After the period of bed care the patient should spend a week or two in the process of getting out of bed, sitting up in a chair for a short while, then gradually increasing the period each day. There is a growing tendency amongst some physicians, notably Dock and Harrison, to permit many of these patients to be treated out of bed, in a chair. I am inclined to endorse this view in the mild cases and feel ready to permit greater activity of the legs in all cases than was the custom formerly. When the patient becomes ambulatory, return to activities should be gradual and in most cases it will be desirable to urge that he should permanently diminish his activities to some extent.

New methods of therapy will necessarily be proposed in the future. Attempts will be made to prevent further thrombosis of vessels. Mural thromboses of the ventricular cavities with subsequent arterial emboli are the cause of some fatalities. One not infrequently sees patients survive the first week of the attack, then while everything seems to be progressing satisfactorily a sudden cerebral embolus occurs with hemiplegia that proves fatal. In such cases if the secondary mural thrombus in the left ventricle could have been prevented from forming during the first several days after the onset of the attack, fatalities would not have occurred. These thrombi are found in about one-third to one-fourth of the patients dying in the acute attack and therefore form a considerable group. One wonders whether or not routine administration of dicoumarin orally during the first week of treatment would prevent the formation of thrombi. The dicoumarin can be given as follows: 5 milligrams per kilogram of body weight for the first dose and then 1.5 milligrams per kilogram daily thereafter. The prothrombin time of the blood must be followed during this therapy. A more effective but also more difficult procedure is to give heparin intravenously or intramuscularly continuously for the first five to seven days keeping the coagulation time about twenty minutes. Although I have tried both



of these methods, the experience is too limited to permit any final conclusions.

One of the most common and harmful errors in the management of acute coronary thrombosis is to outline a lengthy period of invalidism and convalescence. There are too many patients who are told to stay in bed three to six months or to spend a year away from work, "taking it easy." When there is congestive failure, there may be no other choice, but in the absence of congestion, even if chest pain continues, such long invalidism is not only fruitless but often harmful. It generally will be found that the degree of pain will be no greater if the patient is ambulatory. What is more aimless is to advise restricted activities, or as is frequently done, confinement in bed, merely because of weakness. After the first two months, such weakness will not be helped by a program of inactivity. The entire illness, coupled with the alarming precautions taken by the physician and family, produces a state of fear, which often results in a profound neurasthenia and depression. Considerable weakness is necessarily part of the illness itself. This is further accentuated by the immobility during the early weeks in bed. To this is then added a neurasthenic state that finally may condemn the individual to a permanent useless existence. Convalescence

Sometimes these prolonged periods of "convalescence" are the result of insurance considerations, for "total permanent disability" does not begin to take effect until an illness has lasted over three months. At times one is led to believe that this type of insurance has done more harm than good for it has destroyed ambition and encouraged invalidism. What is needed to help this weakness is to encourage the patient to increase his activities and to assure him that his heart has recovered sufficiently to do more. If this point of view is stressed early in the illness, much unnecessary invalidism will be prevented. Many patients ought to be doing part-time work in two months and the majority in three to four months. What has been said about prolonged bed care for patients with coronary thrombosis applies to some extent to patients with other types of chronic heart disease and to those with many other non-cardiac conditions. Not only is prolonged bed rest frequently unnecessary but it may be conducive to the development of further complications such as hypostatic pneumonia, prostatic obstruction, renal stone formation and phlebitis with pulmonary embolism.



## HYPERTENSIVE HEART DISEASE, ARTERIOSCLEROSIS, "CHRONIC MYOCARDITIS," AND RARE FORMS OF HEART DISEASE

### HYPERTENSIVE HEART DISEASE AND ARTERIOSCLEROSIS

HYPERTENSION has a most important bearing on the subject of heart disease. As a concomitant factor in heart failure it probably surpasses all others, so that in most general surveys, hypertensive heart disease heads the list of disabling forms of heart failure. The exact role that is played by the actual elevation of the blood pressure is by no means clear, for so often the same degree of hypertension is well tolerated for many years by one patient and results in severe cardiac insufficiency in another. Even the causes of hypertension are difficult to understand for it seems to accompany a variety of conditions. How much can be explained on the basis of permanent structural changes within the body, especially the arteries, and how much on the basis of an altered nervous or functional state always requires consideration. The marked and even sudden changes that have been observed in the level of the blood pressure in many individuals have necessarily resulted in the firm conviction that the emotions, the nervous system, the endocrine glands and the vasomotor apparatus are all intimately related to this problem.

At the outset it must be recalled that there are a few distinct clinical conditions which, although not really related to the general problem of essential hypertension, produce or are accompanied by some elevation in blood pressure. It is of some importance to be familiar with these conditions for in so far as they exist, physical examination will reveal certain findings that will explain why these particular individuals are hypertensive. In coarctation of the aorta (Chapter 11), for example, there is hypertension in the arms although the pressure in the legs is low. Here the elevation in the blood pressure that is found on routine examination is directly due to a structural abnormality. Similarly one frequently finds hypertension in patients with prostatic obstruction. In some, the pressure will remain elevated until the obstruction is relieved, *e.g.*, by an indwelling catheter, and then it will fall considerably. Such a fall of the pressure has been observed when all other factors, such as rest in bed, have been adequately controlled. At times, this fall may be excessive and it will then be necessary to temporize and delay the prospective prostatectomy until a partial recovery of the blood pressure has taken place. There are many instances in which the eventual prostatectomy produces a permanent lowering of the blood pressure.



Other conditions that are associated with and in some way productive of hypertension are polycystic kidneys, eclampsia, tumors of the adrenals, pituitary basophilism, occasional instances of sudden intracranial hemorrhage, acute nephritis and polycythemia. Hyperthyroidism is also commonly associated with hypertension but the level of the blood pressure falls little if any after the basal metabolism is brought to normal by operation. In addition, with certain cardiac disorders there is an elevation of the pressure which is the result rather than the cause of the disturbance in the heart. When complete heart block develops, the systolic pressure is apt to rise and the diastolic to fall purely as a result of the very slow heart rate. Somewhat similar changes follow the development of free aortic regurgitation. Finally, when hypertension accompanies congestive heart failure, more often than not the pressure level falls as improvement takes place on rest in bed and digitalis. After a considerable experience I feel convinced that some of the fall in blood pressure is a direct result of cardiac therapy and disappearance of dyspnea and congestion and not to be accounted for on the basis of psychic influences or rest in bed. In fact, if the blood pressure does not fall while treating a patient with hypertensive heart failure, it generally indicates that improvement in the heart is not taking place or will not be as great as when the pressure does fall. This relationship is mentioned because there still prevails the idea that when the heart improves it manifests this improvement by an increase in blood pressure. Although this is often the case if the blood pressure is very low to begin with, it is not the general rule when congestive failure takes place with hypertension.

There is one more peculiarity of the blood pressure in relation to heart disease that merits consideration. There is a group of patients having hypertension and angina pectoris in which, following an attack of coronary thrombosis, the blood pressure falls with a complete or partial disappearance of the anginal attacks. When such patients become ambulatory some will thereafter have a low blood pressure, despite the fact that their general health remains even better than before the attack of coronary thrombosis, as they no longer have anginal spells. Under these circumstances a permanent reduction in the blood pressure has taken place following an injury to the heart. This cannot be explained on the basis of structural alterations in the peripheral vascular tree. It seems more reasonable to assume that the previous elevation in the blood pressure was caused reflexly from an irritable focus in the heart and after the coronary thrombosis, the local cause was removed. These experiences and those cited in the previous paragraph, in which it seems that digitalis by improving the heart may diminish the blood pressure, make one suspect that apart from all other causes hypertension in some cases may have its origin reflexly in the heart.

Apart from the conditions just mentioned, hypertension is regarded as an accompaniment of chronic nephritis or pyelonephritis, or as an



idiopathic or essential hypertension. The latter type is most common. One suspects that to a large extent or at least in its early stages it is functional or neurogenic and not based on irreversible structural disease. One cannot explain the oscillations that the blood pressure level undergoes in some individuals on the basis of organic disease. I recall seeing a woman fifty-five years old whose pressure was known to be over 200 mm. for some years. After three weeks' rest in bed there was a steady gradual decline to 140 mm. from the initial level of 240 mm. Thirty minutes after reading a letter containing very distressing news the level immediately jumped 100 mm. back to the original figures of 240 mm. Such changes cannot be explained except as a result of nervous or humoral influences.

The experimental work of Goldblatt has shown that the production of renal ischemia by constricting the renal artery causes maintained hypertension in animals. It has also been shown that, even after such hypertension has lasted many months, removal of the ischemic kidney restores the blood pressure to normal. Furthermore, this type of hypertension was found to be independent of the sympathetic nervous system. The inference from this work is that some product is liberated from the abnormal kidney which circulates in the blood and causes hypertension. This concept appears to be of great promise, affords a clearer understanding of the entire question of hypertension, and may possibly lead to practical therapeutic advances. In fact, already in a few cases of unilateral nephrectomy for hypertension and chronic pyelonephritis the blood pressure has returned to normal.

**Etiologic Factors.**—Among the few known etiologic factors the most important is heredity. There are families with strong tendencies to hypertension and others which tend to hypotension. It has impressed me that marked freckling of the back of the forearms is unusually common in hypertensives and probably denotes a vascular vulnerability. The young nervous hypertensive or potential hypertensive often has a slight fever ( $99.6^{\circ}$  F.). The females predominate over the males in the proportion of about three to two. The menopause seems to be a common period in life in which these changes become prominent although earlier evidences of this tendency are generally available. The hypertensive person will often manifest certain stigmata of neurovascular vulnerability in earlier life. Nosebleeds, menstrual disturbances, flushing—especially of the neck—migraine, palpitation and nervousness will frequently be found. Furthermore, it will be noted that they often have a tendency to slight elevation in the blood pressure on emotional provocation years before they become permanently hypertensive. An insurance examination or a visit to a consultant may raise the pressure to a higher level than the one found by the patient's family physician. In fact, the one who is not prone to hypertension is not apt to show this rise even on nervous tension. It seems, therefore, that the pressure is unusually labile in these vulnerable individuals and that they have



transient hypertension for years before they develop what one might call permanent hypertension.

Although hypertension and arteriosclerosis are frequently associated, it is unlikely that the latter is the cause of the former. One may see extensive sclerosis of the large arteries without hypertension and very little arteriosclerosis with marked hypertension. It is more plausible that prolonged hypertension eventually leads to sclerosis of the arteries, but that during the early years spasm of vessels otherwise sound is going on. The fact that the first portion of the pulmonary artery is rarely sclerosed except in cases of mitral stenosis or other conditions in which pulmonary pressure is elevated, lends support to the role hypertension may play in producing arteriosclerosis. When the blood pressure has been elevated for any considerable length of time, although the radial and brachial arteries may not show much evidence of sclerosis, changes in the retinal arteries will almost always be present. Retinal arteriosclerosis can be judged from the irregularity of the caliber of the arteries on ophthalmoscopic examination and the nicking of the veins at the points where they are crossed by the arteries. When the process is more advanced, hemorrhages, exudate and papilledema may be found.

There are a few diseases that, in so far as they may affect blood vessels, may eventually predispose to early hypertension, *e.g.*, typhoid fever, syphilis, chronic lead poisoning, gout and rheumatic fever. There is some suspicion that this last disease may be a more frequent precursor of hypertension than has been supposed. There is no evidence that foci of infection in teeth or tonsils and the like are of any importance in this question. In the great majority of cases of hypertension, however, none of these infections has been responsible and for the present at least they are best regarded as "essential or idopathic."

**Effect of Arteriosclerosis on the Heart.**—Before discussing hypertensive heart disease the possible effect of arteriosclerosis on the heart must be taken up. The term "arteriosclerotic heart disease" has led to much confusion. In the minds of some this term signifies heart failure resulting from peripheral sclerosis and in others it means coronary arteriosclerosis. If it merely includes those cases in which there is arteriosclerosis of the coronary arteries then it should be called by its proper name, *i.e.*, coronary artery disease. If the former idea is held then it is a misnomer, for there is little if any evidence to show that sclerosis of the peripheral arteries has any appreciable effect on the efficiency of the heart. A study carried out at the Peter Bent Brigham Hospital analyzing all the postmortem material in which there was a high degree of peripheral arteriosclerosis showed that when those cases were omitted in which there was hypertension, significant coronary artery disease or other obvious causes of cardiac disability, such as valvular disease, in the remainder there was neither clinical nor pathological evidence of heart disease. The death of these patients was



due to some surgical or non-cardiac cause. In a word, it was found that even an extreme degree of peripheral arteriosclerosis had no deleterious effect on the heart. It is suggested, therefore, that the term "arteriosclerotic heart disease" be given up.

**Blood Pressure Determination.**—A word about the actual taking of the blood pressure seems worth mentioning. After the patient has relaxed, with the arm in a comfortable position, the pressure is increased above the expected systolic level. The mercury or pressure should be permitted to fall slowly while ausculting below the cuff. Many physicians do this too rapidly and inaccuracies of 20 mm. or more may result. Furthermore, it is important that the first time a patient is examined, palpation of the radial pulse should be performed to check the auscultatory determination. If this is not done one may occasionally start the examination with the pressure level at the "auscultatory gap," at which point no sounds are heard and yet the true reading may be 40 to 60 mm. higher. It is obvious that such an error can easily be avoided, for the radial pulse would be palpable even in this silent zone and the observer would have had to increase the pressure and then would have found that sounds return. Although the auscultatory method is more satisfactory than the palpatory, the latter at times will obviate mistakes. I recall a case in which the physician remarked that he could not obtain the blood pressure. It was found that the diastolic blood pressure in this young girl was 180 mm. and the physician always started his readings at about 150 to 160 mm. If he had felt of the radial pulse he would have known that the blood pressure must have been higher, for the pulse had not been obliterated. Finally, there are instances in which a violent or hyperdynamic pulse, such as is seen in aortic regurgitation, produces a shock and noise with each beat as it strikes the cuff and this is audible in the antecubital space. It may give a falsely elevated systolic blood pressure. In such a case palpation of the radial pulse will indicate the systolic level. I have seen an instance of this type where the actual systolic reading was 140 mm. when it had been read as 250 mm. In this case a definite sound could be heard with the pressure over 300 mm. but it was an impact transmitted from the top of the cuff.

**Clinical Course.**—In following the clinical course of a patient with hypertension there are several definite things for the physician to bear in mind. The exact level of the blood pressure is by no means the important criterion of the patient's progress. The pressure or the associated arterial disease will produce clinical disease in one of several ways. Of greatest importance is what is going on in the heart. This effect may become manifest in one of two ways. Angina pectoris or coronary thrombosis may result from involvement of the coronary arteries or there may develop congestive heart failure, especially left ventricular failure with paroxysmal dyspnea. Next in importance is the possibility of cerebral hemorrhage. There is no method that enables one to predict which hypertensive will develop a cerebral hemorrhage



or when it may be expected. The current opinion is that those with high diastolic pressures are most prone to cerebral accidents. I have had the suspicion that the reverse is true. It has seemed that most patients who develop a sudden hemiplegia have had pressure levels of about 200 to 240 mm. systolic and 100 to 120 mm. diastolic. Those with diastolic readings of 140 to 160 mm. are more apt to have general encephalopathy, cardiac or renal failure but not an outspoken cerebral hemorrhage. May not the high pulse pressure be the more important factor in causing rupture of a large cerebral vessel?

Another group will begin to show significant disease of the kidneys and present the picture of chronic vascular nephritis, eventually developing uremia. A smaller number will have disabilities due to arterial disease of the legs, suffer intermittent claudication or even arteriosclerotic gangrene. Finally there are some in whom the arteriosclerosis of the abdominal vessels is productive of symptoms. Many ill-defined complaints, *i.e.*, indigestion and abdominal pain, are no doubt due to arteriosclerosis of the mesenteric vessels. In this same connection the mild diabetes that is so common in elderly hypertensives may well be due to arteriosclerotic changes in the pancreatic vessels. Frequently the same patient with hypertension may show several of these evidences of arteriosclerosis. He may have a mild glycosuria, slight diminution in renal function and definite intermittent claudication. Another may have outspoken angina pectoris, no renal impairment and more marked sclerosis of the arteries of the legs. Finally, extensive arteriosclerosis may be present without hypertension. It is the function of the physician to try to estimate which is the major route that the vascular degeneration is taking and in general it will be found that the cardiac complications are the most important.

Exactly when the heart will begin to fail in an individual with hypertension is a very variable matter. Neither the level nor the duration of the elevation in the blood pressure can entirely account for the development of cardiac insufficiency. In many instances it is clear that the main determining factor is the integrity of the coronary arteries. Atheromatous changes and narrowing of these vessels can account for the development of cardiac embarrassment in some individuals with only a moderate hypertension. But there are other instances in which the gross appearance of the coronary arteries is essentially normal and yet the heart muscle fails. This unknown factor accounts for much that determines the whole question of congestive heart failure. I suspect that this factor "X" is linked up with the finer vascular bed of the coronary system. It is well known that throughout the human body there are a great many more capillaries than are being used under ordinary circumstances. A skeletal muscle or a glomerulus of the kidneys contains ten times as many capillaries as are functioning at any particular time. The others are closed and are resting for the moment. Under special circumstances the number increases, the walls are opened and



blood corpuscles begin to flow through. This constitutes the reserve function of the organ and may not "cardiac reserve" depend on this very ability to open up new capillaries? When the heart muscle fails and yet shows no obstruction in the main coronary arteries may not the cause lie in some structural or functional defect that prevents the opening of these terminal reserve channels? It is conceivable that if these abnormalities do exist, they might yet be overlooked by the methods of pathological study that are customarily employed in post-mortem examination.

In the course of time an individual with hypertension may develop cardiac failure. In some this may not occur until twenty years have elapsed, in others the downhill path begins after a short time. The heart generally becomes enlarged but the degree of hypertrophy varies considerably in different cases and is not directly proportional to the height or duration of the hypertension. What concerns us here is the development of congestive heart failure and not of angina pectoris. The latter condition can appear any time and suddenly alter the clinical course that the particular case might otherwise have taken. Most commonly, in the former, the first symptom is breathlessness. This appears at the outset on a degree of effort that formerly was well tolerated. Dyspnea on walking is, therefore, the most frequent early evidence of myocardial insufficiency. In many cases unwonted fatigue may have preceded this but because of the numerous non-cardiac causes of fatigue it does not serve as well to identify the condition as heart failure. There is a considerable group of hypertensives in which the shortness of breath appears suddenly, especially at night and may even take the form of acute pulmonary edema. The term "cardiac asthma" is often used to classify these cases. The term is objectionable because the word asthma has too closely associated with it a bronchial condition that has nothing to do with the heart and is comparatively benign. It is better to designate the condition by such terms as "paroxysmal cardiac dyspnea," "nocturnal dyspnea" or "acute pulmonary edema" just as the circumstances warrant.

When dyspnea develops, either of the gradual or of the acute form, it indicates failure of the left ventricle. Examination often shows cardiac enlargement in addition to the elevation in blood pressure. The rhythm of the heart may be perfectly regular or arrhythmias due to extrasystoles or auricular fibrillation may be present. When the auricles are not fibrillating a gallop rhythm and pulsus alternans are frequently present. These two signs are so common and so important in hypertensive heart disease that they should always be looked for. The former is detected by careful auscultation over the precordium where the sounds have a peculiar quality resembling a canter due to an extra third heart sound in diastole. The latter is detected by palpation of the radial artery or better still on determining the blood pressure. While auscultation is carried out below the blood pressure cuff, just as the first sounds



become audible, the pressure should be prevented from falling for a moment and the sounds will be heard to alternate in intensity. This method is even more helpful than palpating the radial artery. Occasionally the same mechanism of alternation of the ventricles will produce an alternating intensity of the heart sounds, murmurs or the apex impulse over the precordium. Similarly the gallop rhythm may result in a bifid apex impulse that can be seen and felt. These two signs will not be found if auricular fibrillation is present.

**Gallop Rhythm.**—It is appropriate at this point to discuss the clinical significance of gallop rhythm. This auscultatory finding is most important, can easily be detected but is often overlooked. The extra sound is generally heard in diastole, *i.e.*, between the second and first heart sounds at the apex (Fig. 150). When the extra sound follows the second sound it is called protodiastolic, when it just precedes the first sound it is presystolic and when it occurs in mid-diastole it is called mesodiastolic. Such terminology is difficult and unnecessary from a clinical point of view. Gallop rhythm occurs most commonly in hypertensive heart disease and coronary artery disease and less frequently in rheumatic valvular disease. Occasionally it is present during acute infections such as diphtheria and rheumatic carditis. Because it is hardly ever associated with auricular fibrillation, the contraction of the auricles must have something to do with the production of the gallop. In fact, I have seen instances in which a typical diastolic gallop disappeared with the onset of auricular fibrillation and reappeared with the return of regular rhythm. In dogs, gallop rhythm has been found to be related to increased pressure in the left auricle. In the non-valvular cases in which gallop rhythm is manifest bundle branch block is also often present. It almost always denotes a fairly serious affection of the heart muscle, carrying with it a life expectancy of a year or two, although occasionally such patients do live longer. The third sound of a gallop rhythm must be distinguished from a normal sound heard in mid-diastole in some healthy hearts. This is not generally difficult for the rate is slow in the latter and somewhat accelerated in the former. Furthermore, patients with a normal third heart sound are apt to be young and show no other evidence of heart disease. Another confusion may arise when a patient who is developing mitral stenosis first shows a third sound in diastole. This may resemble a gallop and subsequently it will be found that the extra sound in fact was the early manifestation of what later proved to be the mid-diastolic or presystolic murmur of mitral stenosis. A gallop rhythm in a patient with rheumatic carditis may be indicative of delayed conduction of impulses from auricles to ventricles (increased P-R interval). This finding may enable the examiner to predict that there is a conduction defect. The prognosis of the gallop need not be grave when it is present in young rheumatic cases. Even the ordinary gallop may disappear when the clinical condition improves, especially when it occurs in conjunction with acute coronary thrombosis or if



the heart rate slows, for it is rarely present when the rate is around seventy.

Finally a word must be mentioned about another type of gallop rhythm. When the extra sound lies between the first and second heart sounds it is called a normal midsystolic gallop (Fig. 149). It can be distinguished from the serious diastolic gallop by the following procedure. If the stethoscope is moved from the apex to the base of the heart in rhythm with the heart beat it will be found that the extra sound, which occurs in the middle of the three sounds, gradually diminishes in intensity and finally disappears, leaving the two normal first and second heart sounds. The important point is that the midsystolic gallop rhythm is benign and the diastolic is grave.

**Cardiac Murmurs.**—The finding of cardiac murmurs in patients with hypertensive heart disease is inconstant. In many of these patients there are no murmurs. More often a systolic murmur will be heard at the apex or base of the heart. This can be either faint or fairly loud. When there is no additional organic valvular lesion the louder apical systolic murmurs probably result from a relative mitral or tricuspid insufficiency and the aortic systolic murmurs from dilatation of the aorta. The dilatation of the ventricular cavities which sometimes is very marked can readily account for stretching of the rings at the base of the valves even when the leaflets themselves are not particularly diseased. Occasionally even the aortic valve may thereby become incompetent in hypertension and result in the presence of an aortic diastolic murmur. Inasmuch as valvular lesions due to other causes, such as rheumatism or syphilis, may coexist with hypertension the corresponding murmurs from these valvular defects may be found.

**Involvement of Lungs.**—The lungs often show evidence of stasis. In the milder cases a few moist râles will be found at the bases of the lungs. When the degree of heart failure is more marked free fluid in the pleural cavities will develop. In the fulminating cases of acute left ventricular failure a generalized pulmonary edema quickly appears with moist bubbling râles throughout the chest, cough and frothy pink sputum. Such a dramatic storm may come and disappear in an hour or two. With the embarrassed respiration, Cheyne-Stokes breathing is frequently present. This may not be detected in some cases if the patient is not carefully observed while asleep for it may be absent while he is awake. The rest of the examination reveals the changes seen in any patient with heart failure. Curiously enough there are some who have considerable breathlessness and even orthopnea who show no peripheral pitting edema, while others may have all the degrees of congestion up to massive anasarca, marked hepatic congestion and engorgement of the cervical veins. Occasionally as one observes peripheral edema becoming more marked orthopnea becomes less troublesome.

**Electrocardiographic Findings.**—The electrocardiographic findings in hypertensive heart disease are variable. For the most part these



depend on the accompanying pathological changes in the myocardium. In most cases there is left ventricular preponderance or left axis deviation, as a result of the enlargement of the left ventricle. This finding has no clinical significance as it is so frequent in other cardiac conditions and even in normal hearts during the second half of life. Bundle branch block or a spread of the Q-R-S complex indicating intraventricular heart block is commonly present. Inversion of the T waves in Lead I or II or those alterations in the ventricular complex that follow disease of the coronary arteries or infarction of the myocardium are also frequently found. In fact any of the arrhythmias or other abnormalities in the mechanism of the heart beat may be present. These changes merely reflect the damage that has already occurred in the function and structure of the heart.

**Treatment and Prognosis.**—Inasmuch as the treatment of hypertensive heart failure is not unlike that for congestive heart failure from any other cause it will not be taken up here (see Chapter 20). One word may be mentioned about the treatment of an acute attack of paroxysmal dyspnea or acute pulmonary edema. In most cases a hypodermic injection of 0.015 gram ( $\frac{1}{4}$  grain) of morphine sulfate with 0.6 milligram of atropine ( $\frac{1}{160}$  grain) will be sufficient to relieve the attack. Sometimes a phlebotomy of 500 c.c. or the application of tourniquets on the extremities with or without the above medication is necessary and effective. In some cases the intravenous injection of 0.24 to 0.48 gram of aminophyllin may be of great value. This may also be used in the evening as a preventive if nocturnal attacks recur frequently. This emergency treatment should be followed by adequate digitalization and diuretics.

The prognosis in hypertension in general is variable. As is well known, many patients live for ten to twenty years or more with a constantly elevated blood pressure. Once either angina pectoris or congestive heart failure develops the outlook changes. But even then some carry on for years. Many different factors need to be judged and even after a complete study is made it is often difficult to make accurate predictions.

There is a form of hypertension that is called "malignant nephrosclerosis" or "malignant hypertension." It is thought by some to be distinct from the ordinary essential hypertension and by others to be merely an advanced and rapidly developing form of the same process. It is associated with a very high diastolic pressure, the readings often being 250 systolic and 150 diastolic. The course is a rapid downhill one ending fatally in about a year or two. Such patients complain a great deal of headache, may have convulsions and show marked changes on ophthalmoscopic examination including papilledema. These cerebral features are described by the term "hypertensive encephalopathy" and may be confused with tumor of the brain. This disease is not rare in younger individuals under forty and even in those in the twenties and



is not amenable to any satisfactory treatment. It has been of interest to me that whereas essential hypertension in general is much more common in females "malignant hypertension" is more common in males. Do the endocrine glands determine this difference?

The surgical treatment for hypertension is now in the process of being investigated. Many hundreds of patients have already been operated upon, some type of dorso-lumbar sympathectomy having been utilized. The operation devised by Smithwick appears to have certain advantages and has gained some support. It is still too early to arrive at a final judgment, but at present it appears to be successful in an appreciable number of cases and can be recommended in selected individuals. It would be well, however, to perform a routine pyelogram examination in all cases of hypertension, if a complete study is desired, for occasionally unilateral renal disease may be detected by that method only. On rare occasions a unilateral pyelonephritis or aberrant renal artery or adrenal tumor will be unexpectedly found. It must be appreciated that tumors of the adrenal not only cause paroxysmal hypertension but also permanent hypertension. The importance of these unilateral lesions is that, although rare, they may be curable by surgical means.

#### "CHRONIC MYOCARDITIS"

The term "chronic myocarditis" has formerly been used to denote a non-valvular condition associated with heart failure. There has been much discussion concerning this term and many substitutes offered. Objection has been made to the term because myocarditis denotes an inflammation and generally no clinical or pathological evidence of inflammation can be found. At present, however, as a result of the great advances in cardiac diagnosis, many of the cases of so-called "chronic myocarditis" can be more accurately named and what was once a "waste basket" for a variety of conditions need now be reserved for only a rare occasion. As one looks back at the cases so diagnosed in the past one will become aware of several clinical entities that now can be well recognized. Some were instances of masked hyperthyroidism that were overlooked. Others were instances of "cor pulmonale" or heart failure from chronic emphysema or pulmonary arterial disease. Still others were cases in which the heart failure was due to avitaminosis (the so-called "beri-beri heart"), or in which edema was present because of a low protein content of the blood. A few were cases in which there was actual valvular disease, either mitral or more frequently aortic stenosis, in which the clinical signs were not elicited or were misinterpreted. I know this has frequently happened in the past because subsequent examination would uncover a systolic thrill at the base of the heart or x-ray examination later would disclose calcification of one of the valves and finally because in some there was marked valvular disease on postmortem examination. A large group of cases that formerly were called chronic myocarditis we now can recognize clinically as



those of coronary artery sclerosis or coronary thrombosis. The increased interest in angina pectoris and the great advances in electrocardiography have enabled us to make these anatomical diagnoses in many instances previously overlooked.

Finally there remains the large group that is now called hypertensive heart disease. This does not mean that the mechanism of heart failure that develops in these cases is adequately explained on the basis of the hypertension. The unknown factor "X" mentioned previously still has a bearing on the problem. But we are enabled to classify such cases under the heading of hypertensive heart disease merely because there is or has been hypertension and there is heart failure without any other predictable anatomical abnormality. When all these well-recognized conditions and a few other unimportant ones like syphilis and anemia are carefully sought for and found absent, there still remains a very small group of patients in whom heart failure takes place. There is no hyperthyroidism, emphysema, valvular disease, coronary artery disease or hypertension and yet the heart muscle failed. Such hearts on postmortem examination are generally enlarged and may show an apparently normal myocardium. The present methods of pathological study fail to explain why the heart muscle was inadequate to maintain a normal circulation. In a few there are found pathological changes in the myocardium but no satisfactory cause for such changes. These cases form the small remainder of what was formerly called "chronic myocarditis" and what may better be termed "non-valvular heart disease" or "chronic myocardial insufficiency."

### RARE FORMS OF HEART DISEASE

**Myxedema Heart.**—With prolonged advanced myxedema certain changes take place in the circulation that are designated as "myxedema heart." Such patients not only have the customary findings, such as a low metabolism, dry skin, coarse, scanty hair, a feeling of coldness, puffy appearance of the face, anemia and a high blood cholesterol, but show more than the usual edema of the legs and marked dilatation of the heart. In some cases what is thought to be cardiac dilatation turns out to be pericardial effusion. Rarely there may be fluid in the abdominal or pleural cavities. In most cases there actually is no true congestive failure despite the cardiac dilatation. Such patients complain of weakness and dyspnea but not of orthopnea. The electrocardiograms show ventricular complexes of low amplitude (Fig. 102). The diagnosis is readily made keeping the above features in mind.

The important point regarding treatment is that patients with this condition do not respond to digitalis but can be cured by thyroid administration. Thyroid medication should be given slowly and cautiously. Too rapid increase in the metabolic rate can bring on acute left ventricular failure or angina pectoris. A dose of  $\frac{1}{2}$  to 1 grain of thyroid gland extract administered daily is often sufficient. If anginal pain



develops it may be best to accept only a partial recovery in myxedema permitting the basal metabolic rate to remain about -10 per cent. Under skilful management the heart returns to normal size, the electrocardiograms show normal tracings and all symptoms disappear.

**Heart Failure from Arteriovenous Fistula.**—There is one form of heart muscle failure which may properly be discussed in this connection, although it is in no way related to either infection or degeneration, *i.e.*, myocardial failure from a peripheral arteriovenous fistula. Here the heart dilates and dyspnea and congestion may develop purely as a result of the increased work of the heart consequent to the short circuiting of the blood. All evidence of cardiac disease may promptly disappear after surgical removal of the fistula or aneurysm. This condition is generally traumatic in origin and often follows a gunshot or stab wound of a limb. The continuous murmur over the fistula or aneurysm, the pulsating veins distal to the lesion, the high pulse pressure and the prompt slowing of the heart rate that follows compression of the fistula are diagnostic of this condition. It is one of the few types of heart failure from purely mechanical causes that is readily amenable to curative treatment.

Not very long ago I learned of a case in which a non-traumatic arteriovenous fistula developed in the pelvis of a kidney. The patient had had advanced heart failure for years and was bedridden. Her physician, Dr. F. B. Camp of Missouri, made the diagnosis by hearing a continuous murmur over the loin. Even after being refractory to all ordinary medical treatment, the patient was cured by a nephrectomy.

**Ruptured Valves of the Heart.**—Occasionally one sees instances of rupture of the valves. In most cases this occurs in previously diseased valves. After a sudden strain a free aortic insufficiency may develop, especially in a pre-existing syphilitic lesion, with a resulting loud or musical diastolic murmur. Without any precipitating effort this complication may arise during the course of bacterial endocarditis. Likewise, in old rheumatic disease of the mitral valve, ruptures may occur, but then they are more likely to take place in the chordae tendineae. This apparently is more frequent than has generally been appreciated, even when no previous disease was present. In such cases, either after some unusual effort or quite spontaneously, a loud apical systolic murmur of mitral insufficiency develops. Cardiac symptoms may appear immediately or insidiously only after some years. Males are more commonly affected than females. All these patients eventually die of congestive failure, although some have survived for months or many years. The possibility of a ruptured valve must always be considered when a loud murmur and cardiac symptoms appear abruptly.

**Beri-beri Heart.**—In the Orient it has long been known that heart failure could result from an inadequate diet. The condition was called "beri-beri heart." One form of this dietary deficiency appeared as peripheral neuritis and the other as congestive heart failure. This condition



is now known to be due to a deficiency of vitamin B. It may take the wet or dry form or be a combination of both. Its occurrence in this country has only recently been emphasized by the studies of Soma Weiss. It is most frequent in chronic alcoholics and in others who have abstained from necessary foods for long periods of time. There is reason to suspect that similar B<sub>1</sub> deficiency with cardiac complications may be present in pregnancy and hyperthyroidism. In the severe form the clinical picture is one of advanced heart failure, both of the right and left ventricular type. The heart is rapid, generally regular, but occasionally shows a transient auricular fibrillation and often has a gallop rhythm. Dilatation may be considerable and yet complete restoration to a normal size results with recovery. Systolic murmurs are frequent but diastolic murmurs are rare. There may be a pistol shot in the peripheral pulse and an increased pulse pressure. In fact, many of the features resemble those seen in thyrotoxicosis or a-v fistula. There often is considerable peripheral edema, engorged liver and pulmonary congestion. Mural thrombosis of the cardiac chambers with resultant emboli may be present. The electrocardiogram may show a diminution or slight inversion of the T waves in any of the leads, and lengthening of the Q-T interval. The important point is that this condition does not respond to ordinary cardiac therapy or digitalis whereas recovery can be dramatic and complete following the administration of 10 to 20 milligrams of crystalline B<sub>1</sub> intramuscularly three times a day (Fig. 129). The subsequent diet should contain meat, flour and yeast.

**Heart Failure with Acute Nephritis.**—Congestive heart failure with dilatation of the heart and the picture of either left or right ventricular failure occasionally occurs during acute nephritis. These cases will show hypertension, pulmonary râles and peripheral edema, as well as evidence of active nephritis. Electrocardiograms at this time may show changes in the ventricular complexes, especially in the T waves, that superficially resemble those seen with myocardial infarction. A similar situation may develop during the toxemia of pregnancy. The response to cardiac therapy is likely to be satisfactory and when recovery takes place the heart may be expected to be normal or essentially so. Restriction of fluid and salt and occasionally intramuscular injections of 1 to 2 grams of magnesium sulfate may be helpful.

**Cor Pulmonale.**—The right ventricle may fail as a result of increased pressure in the pulmonary circuit. This may occur acutely as following a large pulmonary embolus (acute cor pulmonale), or more slowly as in chronic pulmonary emphysema, silicosis or pulmonary arterial disease (chronic cor pulmonale). These conditions have already been discussed (see Chapters 6 and 16). It must be emphasized, however, that great care is needed to distinguish the breathlessness that results from the pulmonary disease "per se" from that which follows heart failure. The treatment and prognosis of the two states are quite different.

**Scleroderma Heart.**—It is now known that pathological changes in



scleroderma are not confined to the skin and subcutaneous tissues. Cardiac complications occur consisting of disintegration of muscle fibers and extensive replacement by fibrous tissue. This can result in cardiac enlargement and congestive failure.

Auricular fibrillation, gallop rhythm and slight prolongation of the Q-T interval of the electrocardiogram have been noted in different cases. I have seen instances in which because of related findings one case resembled chronic rheumatic heart disease and another thyrocardiac disease. The treatment is the same as that for any ordinary case of heart failure.

**Tumors of the Heart.**—Both malignant and benign tumors of the heart are very rare. Metastatic tumors of the heart are about ten times as common as primary tumors. The diagnosis of cardiac tumors is very difficult and most cases are first recognized at autopsy. Peculiar x-ray silhouettes of the heart and the finding of bloody fluid in the pericardium not otherwise explained may lead one to arrive at a correct antemortem diagnosis. Occasionally auricular flutter or fibrillation, transient or permanent, may be present with tumors of the heart, possibly as a result of involvement of the auricles. There is one type of cardiac tumor that has particular interest. This is a myxoma of the left auricle. It is a benign tumor that may produce signs resembling those of mitral stenosis. Such signs may change from time to time apparently owing to alterations of the position of the tumor which may be pedunculated. The resulting events are similar to those seen in rare cases of ball valve thrombus in mitral stenosis. It is not too much to hope that someone may successfully diagnose this type of tumor and attempt surgical removal.

**Cardiac Complications in Lupus Erythematosus Disseminatus (Libman-Sacks Disease).**—A group of conditions that are closely related, if not different manifestations of the same underlying process, has come to light and has been described under a variety of terms. Although there is still confusion in the terminology and complete lack of knowledge as to the etiology, the conditions we now recognize as lupus erythematosus disseminatus and non-bacterial verrucous endocarditis (Libman-Sacks disease) are known to involve the heart. They are not infrequently associated with a sterile type of pericarditis and may show small non-bacterial vegetations on the valves of the heart. In fact there are also rare instances in which the myocardium is involved as manifested by a prolongation of the P-R interval in the electrocardiogram. This group of diseases appears to be closely related in its pathological findings to rheumatic fever, peri-arteritis nodosa and scleroderma, from which it may be differentiated at times, only with difficulty. It also may be confused with subacute bacterial endocarditis because of many features that are common to both diseases.

It must be appreciated that lupus occurs almost solely in females during the years of menstruation, may run a prolonged course of



months or years, may show no lesions on the face at the start or for long periods during its course and can display cycles of high fever with intervening periods of quiescence. There is generally a leukopenia or at least an absence of leukocytosis and it may be associated with an increase in the globulin content of the blood. The heart may be spared but often is involved, as are other serous linings, resulting in pleuritis, peritonitis, pericarditis or endocarditis. There generally is albuminuria and often pulmonary consolidation. The diagnosis is likely to be overlooked until the characteristic butterfly rash appears over the nose and cheeks. The disease is almost invariably fatal.

At present there is no known effective treatment, although spray x-ray treatment is being tried for cases of lupus erythematosus disseminatus with what appears to be favorable temporary alleviation of symptoms.

**The Heart in Addison's Disease.**—With the discovery of effective methods of treatment for Addison's disease by means of synthetic desoxycorticosterone, cortical extract and sodium chloride, a new type of heart failure is now met with. The ordinary patient with Addison's disease has a small heart, often much smaller than normal. Under this new treatment, the heart returns to a more normal size. In some cases the heart is not able to make the necessary adjustment to the rapid changes in dynamics, and with the increase in blood volume and blood pressure, the retention of salt and undue dilation of the heart, pulmonary edema, peripheral edema and even rapid fatality may result. Therefore, if any of the signs of heart failure appear in a patient under treatment for Addison's disease the medication should be omitted for a while and then reinstituted in smaller doses. It should be appreciated that it is the synthetic desoxycorticosterone, which is the salt retaining factor, and the sodium chloride, rather than a preparation such as "eschatin," which is the whole cortical extract, that causes the above cardiac complications.

**Heart Failure from Deformity of the Chest.**—Marked deformity of the chest, especially following poliomyelitis or tuberculosis of the spine, may eventually lead to heart failure. At first only slight dyspnea on exertion or with change of bodily position may be present. The vital capacity of the lungs becomes considerably diminished, while the residual air increases in proportion. As the condition progresses, less and less oxygenation of the blood takes place. With this there may be attacks of palpitation, weakness, cyanosis, tendency to faintness or unconsciousness, and marked dyspnea. Such patients quickly succumb to pulmonary infections. The heart is not much enlarged, though there is often right ventricular hypertrophy. The rhythm is generally regular and the pulmonary second sound is accentuated. The velocity of blood flow, the venous pressure and the cardiac output per minute are within normal limits. Once major symptoms develop the patient's activities become markedly restricted and the downhill progress does not seem to be influenced by cardiac treatment.



Ordinary cardiac therapy is on the whole ineffective in this type of cardiopulmonary failure. The only hope is preventive treatment. These chest deformities produce harmful results if they occur during the growing years of life. Every effort should be made to prevent or relieve these distortions of the chest when the child is young. The orthopedic surgeon should be called in to do whatever he can in order to lessen the deformity. When cardiac symptoms are already present, exercise should be greatly restricted and respiratory infections avoided as far as possible.



## 8

### THYROID HEART DISEASE

**HYPERTHYROIDISM**, whether due to a diffuse hyperplasia or to a toxic adenoma of the thyroid gland, produces certain disturbances in the circulation. It has been the prevailing opinion that hyperthyroidism alone does not cause permanent structural changes in the heart and that when there is gross evidence of congestive heart failure or angina pectoris, other independent conditions will be found such as hypertension, valvular heart disease or coronary artery disease. A recent review of a considerable number of thyrocardiacs, however, revealed many instances of advanced heart failure in which no cause other than thyrotoxicosis could be found and in which complete recovery occurred after appropriate treatment. Frequently arrhythmias occur, that are very troublesome to the patient, which are purely the result of the toxic thyroid gland and it may be expected that these will disappear if the hyperthyroidism is cured. There are many changes that result from hyperthyroidism, independently of other conditions, which are similar to those seen in ordinary forms of heart disease. Inasmuch as thyroid and other forms of heart disease frequently occur together in the same individual and the evidence of an overactive thyroid gland may be very obscure, it becomes extremely important to be familiar with the methods of diagnosis that serve to differentiate the one from the other. This subject is probably the most important aspect of all heart disease for it comprises the one large group of cases in which the difference between accurate and inaccurate diagnosis and treatment is the difference between chronic invalidism or death and restoration of health and life.

#### SYMPTOMS

We are all familiar with the typical picture of exophthalmic goiter. It is not the purpose here to discuss in detail but rather to briefly



mention these features. The main emphasis is to be directed at the group of cases called "masked thyrocardiacs." At the outset it must be appreciated, just as is true in many diseases, that no single sign or symptom is invariably present but that even in the obscure cases the composite picture is sufficiently suggestive to enable one to suspect the proper diagnosis. The cardinal diagnostic points of hyperthyroidism are exophthalmos, thyroid enlargement, nervousness and palpitation. When exophthalmos and a palpable thyroid are absent, as is the case in this particular group of patients, the other symptoms and signs of an overactive gland become more important because only by constant search into these features will the correct diagnosis be made. It must also be borne in mind that these patients, who are nevertheless suffering from hyperthyroidism, come to the physician complaining of the same symptoms as do other patients with heart disease but without hyperthyroidism. Their primary complaints will be shortness of breath, palpitation, weakness, chest pain, swelling of the legs or abdomen, cough, etc. Inasmuch as they often lack the obvious evidence of thyroid disease and have evident organic heart disease of one form or another they are treated for the latter with little if any success and the former is entirely overlooked.

It is always necessary to have the possibility of masked hyperthyroidism constantly in mind when treating cardiac cases. This is especially true if certain peculiar clues are detected. Of first importance is transient auricular fibrillation. Although this phenomenon occurs in other conditions, of none is it so characteristic and in none does it occur as commonly as in hyperthyroidism. It, therefore, should be the invariable practice to suspect a toxic thyroid gland whenever this arrhythmia is observed even when other forms of heart disease such as mitral stenosis are also present. Undue loss of weight, nervousness, tremor of the fingers and excessive perspiration are frequently not investigated with sufficient care when present in cardiac patients. They are too often attributed to the customary forms of heart disease with which they are frequently associated, whereas in some cases a latent hyperthyroidism proves to be the more important cause. This is particularly so if the loss of weight takes place in the presence of a fair or good appetite. Other common findings in these patients are transient glycosuria and transient periods of mild diarrhea or hyperactivity of the bowels. These patients, therefore, are at times treated for diabetes or for some gastro-intestinal disorder. The diabetes here rarely is of much importance, the patients often stating that they have had to pay very little attention to their diet and that it did not seem to matter very much whether they followed a diet or not. It is also of some interest that whereas the ordinary inactive cardiac generally needs cathartics, those suffering from latent hyperthyroidism as a rule do not.

The general appearance of the patient often has given the first suspicious evidence that a latent hyperthyroidism, long overlooked,



was present. The skin takes on a peculiar appearance, rather difficult to describe, that I have called a salmon-colored hue. It is warm, moist, hyperemic and slightly pigmented. The eyes, in the absence of gross exophthalmos, may show a peculiar stare. There is also a certain quickness of motion, an alertness and even an attractiveness to the behavior of many of these patients that one is not accustomed to find in the ordinary case with heart disease. In fact most patients suffering from heart failure, particularly when more or less bedridden, are sluggish both in mind and body, whereas with the same degree of decompensation those who also have hyperthyroidism often retain this peculiar alertness and comeliness. It is also common to learn that these patients prefer cold to warm weather. In addition many have premature gray hair. Often this has preceded by many years the development of the major circulatory symptoms. Incidentally the appearance of premature, rather striking, grayness of the hair has often been an important clue in the diagnosis of other conditions. Early graying occurs in four main groups of individuals. First there is a group of perfectly normal people who begin to have gray or even white hair in the twenties or thirties. Often they have a family history of a similar tendency. Amongst these will be a considerable number who also have a tendency to premature arteriosclerosis. Then there are three diseases with which it is not infrequently associated, *i.e.*, asthma, hyperthyroidism and pernicious anemia. Although the exact causal relationship is obscure it may turn out that early graying of the hair is due to one common abnormality of the endocrine system. Needless to say, such features will not be ascertained or appreciated unless the physician has already been suspecting the thyroid gland. It is very striking in hospital records how the history and physical findings change after the routine admission notes have been made, when the same observer is told that the patient may have masked hyperthyroidism. Only then do appear the observations that the skin is moist and somewhat pigmented and that the patient has preferred cold weather or that there has been a hyperactivity of the bowels. In other words, one has to seek the evidence, for the patient may only be interested in his breathlessness.

It is to be expected that patients with thyrotoxicosis would be prone to avitaminosis. Even with a normal dietary intake, which is not adequate because loss of weight occurs, increased metabolism demands still greater amounts of vitamins. The result is that deficiencies are common. This may manifest itself in the form of a red sore tongue, skin changes, red palms, etc. Possibly some of the cardiac abnormalities in hyperthyroidism are actually the result of vitamin B<sub>1</sub> deficiency.

The examination of the circulation and especially the heart is of the greatest importance in these cases for not only are there peculiarities to be found that help in arriving at the correct diagnosis, but here are also the abnormalities that are associated with the common forms of



heart disease with which hyperthyroidism is confused. It is necessary to repeat that transient auricular fibrillation is very common, but many patients even develop the permanent form of this irregularity. The character of the heart sounds is very apt to be hyperactive. An accentuated first heart sound is common in mitral stenosis, in anemia, in some cases of hypertension, in cases of nervous or unstable hearts but especially so in hyperthyroidism. With this hyperdynamic contraction, often detectable on fluoroscopic examination, there is a diffuse and snapping apex impulse. The vibration of the chest wall resulting from this hyperactive heart makes the impulse diffuse and often creates the impression that the heart is larger than is actually the case. Furthermore a vibration is set up in the chest wall which on palpation feels like a thrill.

When in addition one appreciates the fact that in many cases of hyperthyroidism there is present a systolic murmur either at the apex or base of the heart, one can readily see how easily this condition may be confused with mitral stenosis. This mistake has often been made by most competent physicians. In hyperthyroidism the x-ray may even show a slight prominence in the region of the left auricle similar to that which occurs in early mitral stenosis, although the prominence is both the result of dilatation of the pulmonary vessels and the left auricle. In both conditions symptoms of cardiac disease exist, auricular fibrillation is common, both have an accentuated first heart sound and a systolic murmur and in both there may be a palpable thrill. To be sure the thrill occurs during diastole in mitral stenosis and with systole in hyperthyroidism, but when the heart rate is rapid it is often impossible to time the palpable thrill with any accuracy. The preconception that the condition ought to be mitral stenosis often leads the observer into the false timing of the thrill as presystolic. The one difference between the two conditions is that no murmur will be heard during diastole in cases of hyperthyroidism. This differential point is of the greatest importance. The difficulty becomes greater still when both conditions exist in the same patient, as not infrequently occurs, for one then finds ample evidence of mitral stenosis and has to rely on other criteria to make the additional diagnosis of hyperthyroidism.

There are further peculiarities of the circulation in hyperthyroidism that require mention. It is often difficult and may be impossible to slow the heart rate by the use of digitalis when auricular fibrillation is present. Ordinarily with auricular fibrillation, especially when there is no fever, one expects a specific slowing to an essentially normal rate when adequate doses of digitalis are administered. In fact when such slowing does not occur one is justified in suspecting that the drug is not of normal potency or that the patient has hyperthyroidism. The failure to obtain the expected slowing of the apex rate of the heart in cases of auricular fibrillation following appropriate digitalis dosage has been, in several instances, the first clue that a masked hyperthyroidism was



present. Slowing is promptly obtained if the basal metabolic rate is reduced or brought to normal by the use of iodide therapy or surgery.

In some puzzling cases the determination of the velocity of blood flow helps to decide whether the cardiac symptoms are due to thyrotoxicosis or not. In most cases of ordinary congestive failure the circulation time is slow, *i.e.*, over twenty seconds. In hyperthyroidism the velocity is apt to be fast, even in the presence of congestive failure. The only other conditions in which the velocity of blood flow is rapid are anemia and fever. If, therefore, a cardiac in failure has a reading of twelve to fifteen seconds it is strong evidence in support of the diagnosis of thyrotoxicosis. One other laboratory test may be helpful, *i.e.*, the cholesterol content of the blood. This is frequently decreased to about 130 to 150 milligrams per 100 c.c. whereas most ordinary cardiacs will show readings of over 200 milligrams.

**Blood Pressure.**—Another aspect of the circulation in hyperthyroidism is the blood pressure. The systolic reading may be normal or slightly elevated. In the cases that are occupying our particular attention at present, the so-called "masked thyrocardiacs," the systolic pressure is frequently elevated. This is due to an independent vascular hypertension for it remains essentially unchanged when the hyperthyroid state is eliminated. What is more characteristic is an increase in the pulse pressure, not as striking but similar to that observed in aortic insufficiency. Readings of 160 mm. systolic and 75 mm. diastolic are not uncommon. There may be other features resembling aortic insufficiency such as a capillary pulse, Corrigan pulse, pistol shot in the femoral artery and even the Duroziez's sign. These are all the accompaniments, if not the result of, the high pulse pressure and the peripheral vascular dilatation. Finally the heart rate itself is generally rapid. At times it is difficult to distinguish the tachycardia of hyperthyroidism from that of neurocirculatory asthenia. One helpful distinction lies in the fact that during sleep the rate in the latter condition is normal while in the former the tachycardia, although less pronounced, will still persist. It must not be expected that a rapid heart will be found in all cases of hyperthyroidism. The rate may be under 80 and even under 70, especially in males, in the presence of a toxic gland. I have repeatedly seen such cases where because of the slow heart rate hyperthyroidism had previously been entirely overlooked.

**Basal Metabolism.**—When the possibility of hyperthyroidism has arisen the basal metabolic rate should be determined. Unless the patient is in great distress this can generally be accomplished satisfactorily. The greater difficulty is to interpret the readings. If it is found to be distinctly below normal an active hyperthyroidism can be ruled out. If the figures range from zero to plus 10 or plus 15 per cent it is extremely unlikely that the gland is toxic. There are rare instances, however, in which an active hyperthyroidism is going on, in the sense that cardiac disturbances take place as a direct result of the thyroid gland, while the



basal metabolic rate is perfectly normal. I know of two patients in whom transient attacks of auricular fibrillation were taking place who had a perfectly normal basal metabolic rate. During the course of the subsequent several months, while the attacks of palpitation were recurring, the rate gradually rose to +20 per cent and later to over +40 per cent. Then, after subtotal thyroidectomy, the metabolic rate fell to normal and the paroxysmal fibrillation disappeared. It is evident in such rare cases that the thyroid was "toxic," as far as its effect on the heart was concerned, at a time when the metabolic rate was normal. May it be that patients such as these actually had an original metabolic rate of -20 per cent  $\pm$  (which many healthy individuals have) and when an elevation of 20 per cent occurred they were already "toxic" and yet showed normal readings?

When the readings are plus 20 per cent to plus 30 per cent, it may be no simple matter to decide whether this slight elevation in rate is due to the thyroid gland or is the result of the cardiac failure itself. Slight to moderate elevation in the metabolic rate has occasionally been found by some observers in cases of cardiac failure. These borderline readings may be very difficult to interpret. When properly conducted and repeated metabolism determinations show an elevation of plus 35 or more, in the absence of fever or leukemia, one can be fairly certain that they indicate hyperthyroidism. There is a small group of cases, especially associated with hypertension, in which even this degree of elevation in the basal metabolism will be associated with a normal thyroid gland. I have seen two patients with aortic stenosis who had readings persistently over +40 per cent and yet had normal thyroid glands when examined grossly and microscopically. Tests need to be repeated since not infrequently the initial elevation disappears without specific medication when a second or third test is made. Finally the specific reduction of the basal metabolic rate following iodine administration can serve as a valuable aid in diagnosis. Properly conducted and properly interpreted this laboratory test proves to be of indispensable value in the diagnosis of many of these obscure thyrocardiacs.

### TREATMENT

The treatment of these patients is on the whole a fairly simple matter. All the procedures customarily employed in the treatment of the common forms of heart disease are applicable here, but in addition an iodine solution is administered. Ordinarily 10 drops of Lugol's solution, three times a day, serves this purpose adequately. If there is auricular fibrillation or congestive heart failure, digitalis should be given in the usual amounts, in addition to the Lugol's solution. In most cases a period of seven to ten days will be required to prepare the patient for a subtotal thyroidectomy. Occasionally because of the severity of the complicating heart disease, a longer period will be



necessary. The diet should be abundant and it is well to add vitamins, especially vitamin B<sub>1</sub>. It is well to continue the preoperative medical treatment as long as there is evidence of a progressive improvement as shown by a fall in the metabolism, slowing of the heart rate, and a diminution in congestion. It is useless to wait for the auricular fibrillation, if present, to disappear or to use quinidine preoperatively. The type of anesthetic does not seem to be of any great importance. Many of my patients were given a general anesthetic although others had local anesthesia. The surgeon should be urged to take out more rather than less of the gland, as too frequently an insufficient amount is removed.

The postoperative care consists of continuing iodine therapy for about two weeks, and whatever other measures the cardiac problem requires. If the rhythm of the heart had been regular before the operation, an attack of transient auricular fibrillation the first day or two after the operation is not uncommon. If there had been persistent auricular fibrillation, in many instances the heart will be found to resume its normal rhythm spontaneously during the first week or two postoperative. The basal metabolic rate should be determined from time to time with the hope that it will be normal within two weeks after the operation. In most cases in which satisfactory results are obtained the metabolic rate is already normal by that time, although in some it is still appreciably elevated only to fall to normal more gradually several weeks later. If the rate remains elevated the operation has not been a success and some of the troublesome features such as auricular fibrillation may persist. If the metabolism has returned to normal and this irregularity is still present one should seek for other causes of auricular fibrillation, especially mitral stenosis. Sometimes the murmur of mitral stenosis or aortic insufficiency may become audible only after the metabolism has been brought to normal after the operation. Contrariwise a systolic murmur, so frequent during active hyperthyroidism, often disappears with a return of the metabolism to normal. On several occasions I have found definite evidence of mitral stenosis which was previously unsuspected in patients who had been operated upon for hyperthyroidism, where auricular fibrillation persisted despite a normal metabolism. If this irregularity has not disappeared by a fortnight after the operation one may give quinidine with the expectation that a normal rhythm will be reestablished. It is advisable to do this, bearing in mind that if mitral stenosis is also present, quinidine therapy will entail the same limitations and same dangers that obtain in cardiac patients without thyroid disease.

The results obtained by the above method of treatment are nothing short of miraculous. Patients previously invalided and apparently hopelessly so have been restored to good health and frequently to a state of complete recovery. When it is appreciated that in many of these patients, those included by the term "masked thyrocardiacs," proper



diagnosis could not even have been made up to a decade or two ago and that they would finally have succumbed to heart failure, one can then fully sense the progress that has been made in recent years. This progress has been the combined result of the pioneer physiological work in bodily metabolism, the wonderful development of surgical technique and finally the careful bedside clinical observations that brought to light this hitherto unrecognized group of thyrocardiacs.

The surgical mortality in these cases since the introduction of the preoperative use of iodine has become almost nil. There is practically no cardiac too sick to be helped or too sick to undergo the operation. Not only is the immediate risk very slight but the improvement obtained is quite lasting. Unlike other forms of chronic heart failure, where the improvement after careful treatment is apt to be only too temporary, here a permanent relief is obtained and the patients often are able to carry on full duty without further medication. To be sure they are left with whatever independent form of heart disease, be it valvular, hypertensive or coronary, which happened to be present, but now the previously embarrassed heart resumes its compensated state and is able to continue in this state for years. In no other group of patients, whose members suffer from chronic intractable heart failure despite good medical management, have the members been able to resume varying degrees of physical activities for as long a time as those in this group under discussion. It is this difference in outlook and in treatment that makes the problem of thyrocardiac disease so important.

There is no better way of crystallizing the above problem than to cite briefly some personal experiences. One of the first patients of this type that I had the good fortune to examine in 1921 was a woman in the sixties who had had progressive hypertensive heart failure for two and one-half years, during the last six months of which she had been bedridden. Despite excellent care under the supervision of competent consultants she had been progressively losing ground. The clues in this case were a history of transient auricular fibrillation progressing into the permanent form of the irregularity, a recurrent glycosuria and the failure of the rapid irregular heart to slow on adequate digitalis dosage. In addition there was a certain quickness of motion that the patient manifested despite the fact that she was orthopneic, showed free fluid in the chest and abdomen and had generalized anasarca. The basal metabolic rate was found to be  $+71$  per cent and eventually, after a subtotal thyroidectomy, all signs of congestion disappeared, the rhythm of the heart became regular spontaneously, the metabolic rate became normal, and during a subsequent period of twelve years she never had return of the heart failure. After a long and useful life she finally developed dyspnea and hypertensive heart failure and died about fourteen years after the operation. Her condition remained long overlooked because of the absence of thyroid enlargement and exophthalmos.



Another dramatic result was obtained in the case of a man sixty years old suffering from angina pectoris. During a period of six years there had been an increasing occurrence of the attacks so that finally they came very frequently while the patient was at rest, even awaking him from sleep every half hour or so. The condition remained refractory to all medical methods of treatment although the attacks were always temporarily relieved by nitroglycerin. In desperation the patient wanted to undergo some form of surgical operation such as cervical sympathectomy or alcohol injection of the dorsal roots to obtain relief. He was in this state when I first saw him. The physical examination revealed no abnormalities. The heart rate ranged between 70 and 80. After a period of observation of a week during which time I was at a loss as to what to do, for he was having forty attacks of angina a day, it suddenly struck me that his skin was somewhat moist and slightly pigmented. He also moved rather quickly and jerkily in bed. This led to the suspicion of masked hyperthyroidism. The basal metabolic rate was found to be +45 per cent and on Lugol's solution it fell to +5 per cent. With this fall came a striking improvement with no attacks at night and only about four during each day. No evidence of an enlarged thyroid gland could be made out by palpation or *x*-ray examination. Despite this a very small adenoma was found and removed from behind the manubrium. This emphasizes the importance of *x*-ray examination in searching for a mediastinal goiter when none can be felt in the neck. Often one can be discovered in this way which would otherwise be overlooked. All attacks of angina disappeared after operation and he was able to resume his normal activities for many years thereafter. He experienced anginal attacks only if he hurried up hill. In other words, the anginal state was actually present but was held in abeyance as long as the metabolism was kept normal.

The following experience illustrates the difficulty of recognizing hyperthyroidism in the presence of mitral stenosis. This man, forty years old, had a history of rheumatic fever and was known to have mitral stenosis. He gradually developed increasing dyspnea and over a period of eighteen months became bedridden with advanced congestive heart failure. His physician and the consultant in attendance had observed that it was impossible to slow the heart rate below 100 even when full doses of digitalis were given. When I saw him it was this rapid irregular heart rate in the face of constant administration of digitalis together with a "salmon-colored skin" that first made me suspect a latent hyperthyroidism. While in the state of advanced congestive heart failure with an apex rate of 170 to 180 the basal metabolic rate was found to be +40 per cent. On the same dose of digitalis (0.1 gram twice daily) but with the addition of 10 drops of Lugol's solution three times a day, the heart rate quickly fell from 180 to about 60, the basal metabolic rate to +6 per cent and there took place an extraordinary diuresis. Within ten days the patient felt perfectly well. It was



believed, however, that the improvement would only be temporary if the toxic gland were not removed. There was no exophthalmos or palpable thyroid gland. It was difficult in this case and in many others to convince the patient that he should undergo an operation. In fact it was not simple in some instances to prevail upon the surgeons to perform the operation, for often they did not believe the gland was diseased. However, after the operation in this case the striking improvement that had taken place on Lugol's solution was maintained despite the fact that the patient still had mitral stenosis and auricular fibrillation. He was able to resume his work which he had been forced to give up two years previously.

One could go on at great length and describe numerous other instances in which apparently hopeless cardiac invalids were resurrected by, first, the detection of suspicious evidence of masked hyperthyroidism and then by the institution of proper treatment. The important lesson from all this is constantly to suspect an undetected toxic thyroid gland when treating patients with cardiac disease. Although a very few such patients will be sufficiently improved by the use of iodine therapy alone, the vast majority will do better to undergo a subtotal thyroidectomy.

The question of *x*-ray treatment for hyperthyroidism deserves a word of comment. I have had little experience with it because it is time-consuming and too often ineffective. The fact that operation is so safe nowadays, makes surgery the method of choice. In a series of ninety-nine patients with thyrocardiac disease, many of whom were critically sick, even still showing congestive failure at the time of operation, there was no surgical mortality whatever. These figures speak for themselves.

One last word is appropriate concerning possible medical treatment in the future. It is quite clear that the fundamental cause of hyperthyroidism does not lie within the thyroid gland. The thyrotropic hormone of the pituitary gland has much to do with the thyrotoxic state. It is also thought that exophthalmos is determined by some control from the pituitary gland. Certainly patients may be cured of hyperthyroidism and yet progress with malignant exophthalmos. In these cases the administration of thyroid gland extract with iodine is indicated. The ultimate solution must be found outside the thyroid gland. It is particularly hopeful to learn that a new compound has been discovered that reduces the basal metabolic rate. Quite recently Astwood has shown that thiouracil given in the form of two to four tablets orally, each containing 0.1 gram, will lower the metabolism in ten to fourteen days in cases of thyrotoxicosis and possibly in cases with a normal gland. Many cases of hyperthyroidism have already been cured by this new drug. I have even seen several instances in which persistent auricular fibrillation, which had lasted for months, reverted to normal rhythm as the metabolism fell to normal. Future work along this line is



awaited with interest. In the meantime care must be exercised in administering thiouracil, as occasionally toxic reactions occur such as agranulocytosis and skin rashes.



## 9

### SYPHILITIC HEART DISEASE

SYPHILIS affects the circulation in one of several ways. There is a very rare condition in which it involves the peripheral blood vessels producing a picture of malignant arteriosclerosis and hypertension. The more important lesions are aortitis and aneurysm, aortic insufficiency, involvement of the coronary arteries and of the myocardium. It is much more common in males than in females and in the colored race than in the white race.

**Aortitis and Aneurysm.**—Although the spirochetes no doubt localize in the aorta a short time after the initial infection, clinical evidence of syphilitic disease of the aorta ordinarily does not become apparent for many years (fifteen to twenty on the average) after the primary sore. However, there are occasional instances in which this does occur within one to two years. The favorite site is the first portion of the ascending aorta although any part may become affected. For many years, therefore, a syphilitic process may be going on in the aorta without any impairment in health or any abnormalities on physical examination. When syphilitic aortitis first becomes detectable it will do so by producing a slight dilatation of the ascending aorta. It will be difficult to elicit this by percussion. *x*-Ray examination will be necessary and even then it is no simple matter to differentiate a luetic dilatation from one that accompanies hypertension or one that is due to arteriosclerosis.

For the most part syphilitic aortitis of itself produces no symptoms. When it does so there is pain in the chest that resembles somewhat that seen in disease of the coronary arteries. The resemblance, however, is only superficial for the pain is not particularly brought on by walking and is not so characteristically constricting in type as it is in angina pectoris. Here the pain is more boring and more steady. It is frequently more troublesome at rest or at night in bed or when the patient lies in certain positions. Physical examination for the most part reveals no essential abnormalities. A localized systolic pulsation may be seen or felt at the base of the heart particularly in the second right interspace. On auscultation there need be no murmurs or irregularities whatever or only a slight systolic murmur is present. The aortic



second sound often takes on a peculiar accentuated or metallic quality. When there are no other more definite evidences of syphilis it is extremely difficult to make a clinical diagnosis. As has just been mentioned the *x*-ray may be very helpful but often still leaves us in doubt. The Wassermann test will be found positive in only about 75 to 85 per cent of the cases so that when negative it cannot satisfactorily rule out the diagnosis and when positive it may not reflect a syphilitic process in the aorta.

When the syphilitic process in the aorta produces a true aneurysm the diagnosis is more readily established. This takes the form of diffuse or sacculated dilatation. When the latter is present one can safely assume that syphilis is the cause. Aneurysm of the aorta produces symptoms mainly by pressure on neighboring structures and this will in a large measure depend on the portion involved and the direction that its enlargement takes. It may press on the trachea or either bronchus and produce cough (often with a brassy quality), a tracheal tug, or atelectasis of the lung. It may expand backward and cause erosion of bone in the vertebrae and ribs. This then becomes quite painful. Or it may erode bone anteriorly through the manubrium and clavicles and present itself as a pulsating tumor in the front of the chest. Inequality of the pupils and pulses or aphonia may result from pressure on nerves or large arteries. Aneurysms may attain a considerable size and produce no noticeable symptoms merely because their growth does not happen to cause pressure on any of these important structures.

Apart from the findings just mentioned an aneurysm often produces local pulsations in unusual sites. Such pulsations are to be sought for by inspection and palpation over the base of the heart and in the upper back between the scapulae. All the points referred to should be gone into when an adult complains of a boring pain in the chest and careful *x*-ray examination should be made. It has been said that there are two types of aneurysm of the aorta—those with signs and those with symptoms. One should add a third, *i.e.*, those with neither signs nor symptoms. I have seen patients with syphilitic aortic aneurysms the size of an orange, when there were no symptoms and no abnormal physical findings (except in the *x*-ray) even after the exact location of the aneurysm was known.

The efficiency of the general circulation is not materially affected by syphilitic aortitis with or without aneurysm. If the valves, the coronary arteries and the myocardium are spared there will be no evidence of congestive heart failure. The largest syphilitic aneurysm I ever saw occurred in a patient who had a perfectly normal heart. There never was evidence of cardiac failure and on postmortem examination the weight of the heart was less than 300 grams and the valves and myocardium were normal in every way. The aneurysm was almost as large as a football and originated from a hole in the aorta 1.5 centimeters in diameter. When congestive heart failure is present with luetic involve-



ment of the aorta some added factor will be found which acts deleteriously on the heart.

The syphilitic process may extend downward and involve the mouths of the coronary arteries which open just at the root of the aorta. This is not a rare complication and when it does occur all the possibilities exist that attend an inadequate coronary circulation. Anginal attacks may occur and sudden unexpected death result. Syphilis, therefore, produces angina pectoris by narrowing the orifices of coronary vessels. It hardly ever involves the main course of the arteries as occurs in ordinary cases of angina. There have been a few cases reported in which the mouths of both the right and left coronary arteries were completely occluded by a gradual syphilitic process. The circulation through the heart had apparently been adequately maintained although no flow through the coronary arteries was possible. The work of Wearn has emphasized the possible role that may be played by the Thebesian vessels in maintaining the health of the heart. Compensatory circulation through these small channels that open directly into the ventricular cavities probably explains how such patients could have carried on in life and may also account for the compensatory mechanism that goes on in non-syphilitic cases of coronary artery disease.

**Aortic Insufficiency.**—Of greatest importance is the effect of the syphilitic process on the aortic valves, as this accounts for most of the disability and mortality in syphilitic heart disease. The disease often extends downward and destroys the integrity of the aortic orifice. It then produces aortic regurgitation just as occurs in rheumatic fever, but unlike the latter it never results in stenosis of the aortic valve. The appearance of the aortic cusps also differs in the two conditions. In the syphilitic form the commissures become separated while in the rheumatic they become fused. When aortic insufficiency is present the heart may become enlarged and in time congestive failure results. Cardiac enlargement does not occur because of the aortitis but as a direct result of the valvular insufficiency. When dyspnea and other evidence of circulatory failure intervene the prognosis is apt to be quite grave as response to treatment is on the whole unsatisfactory.

The diagnosis of syphilitic aortic insufficiency will depend on eliciting evidence of this type of valvular defect (Chapter 4). It is most important to appreciate that during the early stages, the aortic diastolic murmur which is so important may be very faint and only audible with the patient upright and after a forced expiration. At times it is difficult to determine whether aortic insufficiency is part of a syphilitic, rheumatic, hypertensive or arteriosclerotic process and will require all the means available to help in the differentiation. The difficulty may be particularly great because an Austin-Flint murmur resembling mitral stenosis is not rare in syphilitic aortic insufficiency. In general, luetic aortic incompetency occurs at about the age of forty to sixty, is much more common in males than females, is only rarely associated with auricular



fibrillation and never results in stenosis of valves. It is compatible with good general health and vigor for many years but when cardiac decompensation first develops the downward progress from then on is apt to be rapid. Cheyne-Stokes breathing and nocturnal dyspnea are common.

**Syphilitic Myocarditis.**—Direct syphilitic involvement of the heart muscle is very rare. When it does occur it may take one of two forms. There are isolated instances of localized gumma of the heart. When such a process involves the conduction apparatus, complete heart block may result. There are rare instances in which a myocardial gumma becomes calcified or causes a localized aneurysm. Under such circumstances, *x*-ray and kymographic examination may help in diagnosis. The other form is a diffuse luetic myocarditis. This is also quite rare and produces a rapid development of congestive heart failure that fails to respond to the customary methods of treatment. Syphilitic disease of the heart muscle has been regarded by some observers as being quite common. It used to be looked upon as a common cause of angina pectoris especially in younger people. The opinion has gradually changed and now it is thought that syphilis is a rare cause of heart disease apart from its effect on the aorta or the aortic valves.

### TREATMENT

When congestive failure develops from syphilitic heart disease the response to treatment is rather unsatisfactory. The hope, therefore, lies entirely in the preventive aspects of this disease. All the efforts that are being made to diminish the incidence of the primary infection will naturally prevent the subsequent development of this form of heart disease. Likewise the early detection of syphilitic infection and the careful and thorough treatment of syphilis in its early stages will do a great deal to prevent these late complications. In fact it is already apparent, since the introduction of the Wassermann test, the discovery of salvarsan and the present public health campaigns against venereal disease, that syphilitic heart disease is becoming much less prevalent.

If syphilitic cardiovascular disease is detected before either anginal or congestive symptoms have developed treatment is directed in the hope of preventing or delaying such complications. A course of potassium iodide and mercury or bismuth should be given for about one month and followed by arsenic administered intravenously. Ten to 20 drops of saturated solution of potassium iodide may be given three times a day. During the same time some form of mercury such as the succinimide should be given intramuscularly. The dose is 0.013 gram ( $\frac{1}{8}$  grain) three times a week for six doses, then increasing the dose to 0.026 gram ( $\frac{2}{8}$  grain) three times a week. After such a preliminary course it is well to give six weekly intravenous injections of neosalvarsan beginning with 0.2 gram, gradually increasing to 0.45 gram. Instead of mercury or as an adjunct to it bismuth subsalicylate (0.2 gram in



2 c.c. of oil, given intramuscularly once weekly) may be employed. It is not the purpose to push antiluetic treatment to the point of making the Wassermann reaction negative. Although one would welcome this change it often is impossible to change the reaction of the blood, and continued vigorous antiluetic treatment may do more harm than good. After a period of treatment, as outlined above, has been carried out it may be repeated once or twice a year.

When congestive failure is present all the customary methods of treatment are employed such as digitalis, diuretics, etc. (Chapter 20). I have seen practically no evidence that mercury or arsenic has been useful at this stage of the disease. Although I would not hesitate to use potassium iodide and mercury after the state of compensation has been improved I think it is better to refrain from using intravenous arsenical preparations. This is particularly true when the coronary arteries are involved and anginal symptoms are present. I know of two instances in which sudden and unexpected death occurred a few minutes after an intravenous injection of salvarsan. Inasmuch as there is so little good that one might obtain from the intravenous use of arsenic, the possibilities of such catastrophes lead one to the opinion that it should not be employed whenever disease of the coronary arteries is suspected.

When pain is due to disease of the aorta, especially when an aneurysm is present, salvarsan can be very efficacious. This should be given as outlined above after a preliminary course of potassium iodide and mercury. Aortic pain often responds very satisfactorily to this treatment and may even disappear entirely. This pain should be carefully distinguished from anginal pain, for intravenous treatment is to be avoided in the latter condition. If the pain is troublesome and refractory to ordinary treatment alcohol injections of the dorsal roots may be tried.

In a few isolated and properly selected cases of sacculated aneurysm of the aorta, wiring has been performed apparently with some success. The cases in which this operation may be applicable are those in which the heart is essentially normal and the aneurysm both large and localized. The purpose is to produce a firm clot in the aneurysm and thereby prevent or retard its continued enlargement.



## BACTERIAL ENDOCARDITIS

THERE has been considerable confusion in the minds of physicians concerning the term "endocarditis" and its clinical significance. When the valves are involved by acute rheumatic fever there occurs an acute endocarditis which often leads to valvular deformities and eventually to what is called chronic rheumatic valvular disease of the heart. The original endocarditis under these circumstances is non-bacterial. If the heart is examined postmortem at the acute stage of the disease the valves will show only slight alterations. Pinhead vegetations may be found and slight irregularities of the valve margins may be present. Bacteria will be present but rarely in the blood stream or on the valves. In other words, until the actual cause of rheumatic infection is discovered acute rheumatic endocarditis is regarded as a non-bacterial endocarditis. Rheumatic endocarditis is therefore an affection from which clinical recovery takes place in the great majority of cases and as a result of which chronic valvular deformities develop years later which may lead to congestive failure and the other complications of rheumatic valvular disease. The condition which engages our attention in this discussion, on the other hand, is a bacterial affection of the valves which in the past has been almost invariably fatal. Therefore, for clinical purposes, under the term "endocarditis" should be included two forms. The first form is the non-bacterial endocarditis which is mainly rheumatic. The second form is bacterial endocarditis which for purposes that will become evident can be conveniently subdivided into two subgroups, acute and subacute.

## ACUTE BACTERIAL ENDOCARDITIS

Most of the clinical features of acute bacterial endocarditis are similar to those seen in subacute bacterial endocarditis, except that they occur more rapidly, the whole illness is more violent and it lasts a much shorter time. The cause of the brevity and the fulminating nature of the disease is the type of micro-organism that is concerned and the particular disease of which the endocarditis is only a part. The more common organisms responsible for acute bacterial endocarditis are hemolytic streptococci, pneumococci, staphylococci, gonococci and influenza bacilli. There are rare instances in which other bacteria cause this same disease. Recently instances due to undulant fever have been reported. Occasionally an infection with one of the above organisms may run a prolonged course and would rightly belong to the type designated as subacute. This is particularly true of *Bacillus abortus* infection (undulant fever). The subacute type, however, is so common and so distinct that



for practical purposes it deserves to be distinguished from those designated as acute endocarditis.

Acute bacterial endocarditis is a part of an overwhelming general infection with involvement of the blood stream. It is always secondary to some other primary disease process. The patient may have an acute or subacute gonorrheal infection and during a phase of bacteremia, if the valves of the heart become involved, acute bacterial endocarditis due to the gonococcus develops. Another has an ordinary lobar pneumonia, and in a similar fashion a pneumococcus infection of the valves develops. In this way almost any infectious process can at times become the primary cause of involvement of the valves.

It is often difficult and at times impossible to diagnose this condition. The evidence of endocardial involvement is apt to be overshadowed by the severe septicemia and underlying disease. A patient is very ill with pneumonia or with streptococcus septicemia and the fact that the valves are affected may be overlooked unless significant murmurs develop or embolic phenomena occur. These are not invariable, however. There is present a considerable fever with or without chills, rapid pulse and the general appearance of a severe acute infection. The spleen becomes enlarged, petechiae and emboli are common and meningitis is not rare. Many of these features merely indicate a severe infection and do not necessarily point to a disease of the valves. The finding of a positive blood culture establishes the fact that there is a septicemia but does not prove that bacteria are lodged on the valves. This accounts for the fact that many such cases are first detected on postmortem examination.

The present use of sulfa drugs and other potent chemicals such as penicillin will probably result in curing many patients with bacterial endocarditis (formerly fatal) in which the absolute diagnosis could not be or was not established. It would hardly be wise to delay treatment in a suspected case merely because a certain diagnosis had not yet been made or the blood cultures were still sterile. In this way we may now see recovered cases with compensated chronic valvular heart disease, not rheumatic in origin, that we had never seen before.

Unlike subacute bacterial endocarditis, this disease frequently affects valves that were previously normal. Both sides of the heart are vulnerable to this infection. Although the aortic and mitral valves are more commonly involved, the pulmonary and tricuspid are often affected. The disease runs a course of several days to a few weeks and has been almost always fatal. In the past, many chemicals and sera have been used without success. With the discovery of the sulfa drugs some cures have been obtained. The most recent advance is the use of penicillin. Although it is too early to judge, this drug is likely to prove to be the most effective remedy, for already some amazing results have been obtained. It is not unlikely that the newer drugs, if given during the stage of bacteremia before the valves are involved, may actually prevent the de-



velopment of endocarditis. Furthermore, they should be tried even after the diagnosis of acute endocarditis has been established.

It is of some interest that although pneumococcus endocarditis is quite common among fatal cases of pneumonia, it cannot be regarded as a cause of chronic valvular diseases. I have seen only one case in which it seemed that an attack of pneumonia was the cause of a chronic valvular disease. In this instance there were no murmurs or other evidence of heart disease before the illness and definite signs of aortic insufficiency appeared directly after the pneumonia. When the physician is inquiring into the past history of his patients and is told that there was some leak in the heart ever since an attack of pneumonia he has reason to doubt the assumed relationship. On closer scrutiny it may be found that the murmur antedated the attack of pneumonia and was due to a previous rheumatic infection or that the alleged pneumonia was in itself rheumatic in type or actually an instance of rheumatic pericarditis with signs of compression of the lungs which cannot be distinguished from the signs of pneumonia. However, in the future the situation may be different. As some recoveries may now be expected, following the use of the newer drugs, we will undoubtedly see cases of chronic valvular disease resulting from cured instances of bacterial endocarditis.

### SUBACUTE BACTERIAL ENDOCARDITIS

**Predisposing Causes.**—As a result of considerable accumulated experience it has seemed that there are certain individuals who are more prone to develop this disease and others who are much less so. As a rule patients who develop subacute bacterial endocarditis have had some sort of heart murmur for years but otherwise have been fairly well. The heart is only slightly or moderately enlarged, the rhythm is regular and there is no hypertension. They have had little if any dyspnea and have been able to carry on usual activities. When the original infection was rheumatic fever, they are apt to have had one or only two bouts of this, thereafter remaining free from recurrent rheumatism. In other words, those patients with valvular deformities who on the whole are in comparatively good health are most vulnerable. In contrast to this, it is very rare indeed to see the disease develop in patients who have had chronic persistent auricular fibrillation or who have previously had congestive heart failure or hypertension. In fact it is not very common in outspoken cases of mitral stenosis, particularly in those in which there is marked constriction of the valve. It is frequent in patients with well-compensated aortic insufficiency or stenosis or mitral insufficiency. It practically never develops in a previously normal heart, but rather in those which have some abnormality of the valves or endocardium, either rheumatic or congenital in origin. In general it may be said that 20 to 25 per cent of all patients suffering from valvular disease succumb to bacterial endocarditis.

In an analysis of 111 cases of subacute bacterial endocarditis it was



found that a past history of rheumatic fever was obtained in forty-two instances, of chorea in three, and of both in six cases. There were eleven other patients who had a history of scarlet fever. There is an indirect association between scarlet fever and rheumatic fever in that the latter may follow in the wake of the former. Some patients who develop a heart murmur after an attack of scarlet fever will be found on close scrutiny to have had mild limb or joint pains during their convalescence and it can be surmised that they actually had an attack of mild rheumatic fever which was precipitated by the scarlatina infection. Recently it has been found that those children who had cardiac involvement supposedly as a result of an attack of scarlet fever have had the same high familial incidence of rheumatic heart disease as occurred in ordinary cases of rheumatic fever. This leads one to believe that scarlet fever produces a chronic endocarditis only in those susceptible individuals who are already potentially rheumatic. Of the entire group of 111 cases in only one could no other cause but a luetic aortic insufficiency be found to account for the previous valvular injury. There were eight other cases in which there was a positive Wassermann reaction in the blood but in all there was stenosis of the aortic or mitral valve or a past history of rheumatic fever which made it likely that a rheumatic rather than a syphilitic lesion was the predisposing cause. Congenital lesions were the site of the bacterial endocarditis in five cases.

The time elapsing between the original injury to the valve and the development of bacterial heart disease is difficult to ascertain accurately. In a group of sixty cases the average interval between the first time the patients knew they had "heart disease" and this final disease was about twelve years. The shortest interval was one year and the longest was forty-five years.

**Incidence.**—Males predominate over females in the proportion of three to two despite the fact that among all cases of mitral stenosis the proportion is two to one in favor of females. This reflects the relative antagonism between mitral stenosis and subacute bacterial endocarditis. The most prevalent decade is twenty to twenty-nine, although the disease occurs in individuals at all ages from childhood to old age. It is curious that although males strongly predominate after the age of thirty the reverse is true under the age of twenty.

**Clinical Features.**—Subacute bacterial endocarditis or endocarditis lenta is a fairly common condition and is mainly due to the streptococcus viridans. The infection becomes implanted almost invariably on previously injured valves. Generally there has been some rheumatic valvulitis from which a satisfactory recovery had taken place or much less frequently there has been some congenital defect like patent ductus arteriosus, ventricular septal defect or bicuspid aortic valves. Even when evidence of an early rheumatic infection cannot be elicited it will be found that some sort of heart murmur was present before this final infection developed.



The onset of the disease is rather gradual. It generally is initiated by a so-called "simple cold" or sore throat or not infrequently by the extraction of a tooth or the removal of tonsils. Rarely a local septic infection such as is seen after a simple abrasion of the skin may be the precipitating cause. It may occur postpartum or postoperatively if there has been some minor secondary infection. When these causes usher in the disease it must be borne in mind that if the heart were originally in a normal state subacute bacterial endocarditis would not have developed. It is because the patient already has some old abnormality of the endocardium and especially if he belongs to the vulnerable group, that a few stray bacteria in the blood stream start growing on the valves. Very frequently the onset is grippelike or may resemble typhoid fever. The patient may have complained of a "cold" and state that since then he has not felt well. Malaise, anorexia, sweats and chills gradually develop. There is a slow loss of strength and weight, although during the early few weeks many are able to do and some actually continue at their usual work. Fever is practically always present and generally is of the swinging type, about  $98^{\circ}$  to  $99^{\circ}$  F. in the morning and  $101^{\circ}$  to  $103^{\circ}$  F. in the evening. Occasionally the fever is very slight, hardly rising above  $99^{\circ}$  F. for some time.

The various important features of this disease occur in no constant order. In some, only the symptoms listed above develop for a long time; in others, complications that ordinarily come late may be the initial event calling attention to the gravity of the situation. I recall an instance in which a young dentist suddenly developed blindness in one eye due to a retinal embolus. Although he had not felt exactly well for a week or two, he was at work when the visual accident occurred. The significant points to bear in mind are petechiae, splenic enlargement, red blood cells in the urine, clubbing of the fingers, painful finger tips, emboli and a positive blood culture. These are particularly important because they help to distinguish this disease from an active rheumatic infection of the heart or its valves.

The petechiae are small oval hemorrhagic areas about 1 to 2 mm. in length which on close inspection will show a gray or white center. They are commonly found in the conjunctival sac, the mucous membrane of the mouth or on each side of the neck, although they may occur anywhere over the body. They are almost pathognomonic of a bacterial endocarditis. The spleen gradually becomes enlarged so that at first the change may be suspected by percussion and finally by palpation. The enlargement is due both to embolic infarctions and to the general septic process. A palpable spleen is rarely found in rheumatic fever itself or in valvular disease of the heart even when there is congestive heart failure. The liver often becomes markedly enlarged during heart failure, but not so the spleen. When the abdominal viscera show evidence of passive congestion splenic enlargement hardly ever



reaches the point at which this organ becomes palpable on abdominal examination.

The finding of a considerable number of red blood cells in the urine is also distinctive because it does not occur in ordinary heart disease unless there is an active nephritis as well, and only very rarely during simple rheumatic fever. Acute pericarditis is common with rheumatic infections and very rare with bacterial endocarditis. Clubbing of the fingers is common after the first few weeks of this disease. It is not seen in rheumatic fever or in ordinary heart failure. It does occur in congenital heart disease when there is chronic cyanosis, in chronic pulmonary infections and in some other cardiac states associated with cyanosis. Its presence as a constitutional or hereditary condition at times makes its interpretation difficult. Apart from the clubbing, there is also a peculiar type of pain in the finger tips that deserves attention. Patients often complain of sudden pain in the balls of their fingers. It comes, lasts a few days and then completely disappears. During this time one may see a somewhat purplish spot at the tip of the fingers or under the nails which gradually fades entirely. Because of the unusual location, pains of this type are highly distinctive of a bacterial endocarditis. Similar pains and discoloration of the skin can occur elsewhere, of course, but then their interpretation is not as simple, although splinter hemorrhages anywhere in the skin are fairly distinctive.

The recovery of bacteria from the blood stream by cultural methods is the most important evidence of bacterial endocarditis. The ease with which this is obtained varies considerably in different cases and does not necessarily depend on the height of the fever. At times repeated blood cultures may be negative although other aspects of the case indicate a severe infection, whereas in some instances in which the afternoon temperature is less than 100° F. a positive culture is readily obtained. The organism that is found is generally the streptococcus viridans or the non-hemolytic green-producing streptococcus. These bacteria may be slow to grow in artificial media so that the cultures should be saved for a week or two. A positive blood culture is very rare during rheumatic fever and will be found in most cases of subacute bacterial endocarditis if repeated search is made.

Finally one of the characteristic features of the disease is the occurrence of emboli. They are common in the spleen, producing acute sharp pain over the lower lateral aspect of the left chest, in the kidney causing pain in the loin or in the abdomen with radiation to the groins, in the limbs, or in the brain with the necessary consequent complications. Emboli to the intestines sufficient to produce significant symptoms are rare, although occasionally gangrene of the intestines may result. The tissues involved in these various infarctions rarely break down or suppurate. When the vegetations involve the right side of the heart pulmonary emboli occur.

In a small group of cases, as the disease progresses, the active stage,



as expressed by fever and chills, quiets down and a picture of chronic progressive renal insufficiency develops. These patients appear to have chronic nephritis with nitrogen retention and marked anemia. They may have little or no fever for long periods of time, show no bacteria in the blood stream and die a renal death. The clinical diagnosis will then have to rest on the presence of cardiac murmurs, clubbed fingers, anemia and other general peculiarities of the illness.

There is a type of case in which the vegetations may be confined to the right side of the heart and in which peripheral arterial emboli are striking by their absence and the blood cultures prove negative for most of the course of the illness. Small emboli, however, become dislodged and produce multiple pulmonary infarctions. Such cases may resemble pneumonia and be wrongly diagnosed as such. This state of affairs is apt to occur when a bacterial endocarditis develops on the defect of a patent ventricular septum or patent ductus arteriosus. I have seen an instance in which on postmortem examination a ring of vegetations encircled the aperture of the septum on the right ventricle while there were no vegetations in the left ventricle. It seemed that the flow of blood from left to right ventricle prevented the growth of the bacteria against the stream. The presenting symptoms in this case were pulmonary, and the peripheral arterial phenomena were lacking.

The course of the illness is a gradual downhill one. It may be suddenly altered or terminated by peculiar accidents such as rupture of a valve, the ventricular septum or the ventricular wall, or by a gross cerebral embolus. There is even a recorded instance in which an embolus into the coronary artery from a vegetation of the aortic valve occurred with instant death in an individual who was well enough to be playing golf when he was stricken. Ordinarily, however, the disease lasts several months or longer with slow wasting such as one might expect with a prolonged septic process. The symptomatic complaints may be very few for long periods of time. In fact some would hardly think there was anything wrong and wonder why they are regarded as sick and kept in bed. During the early weeks or even for months there may be no dyspnea or evidence of cardiac failure. These do occur later in the disease and heart failure may be regarded as the final cause of death in a considerable portion of cases. Other patients merely waste away with increasing anemia. In a small number a major embolus brings to a close this lingering disease. In a very few the end-result is a glomerular nephritis with uremic manifestations.

**The Valves Involved.**—It is frequent to find on postmortem examination that the vegetations of subacute bacterial endocarditis involve more than one valve. When they are confined to one valve they are almost as common on the mitral as on the aortic leaflets. Much more rarely are the pulmonary or tricuspid valves affected. A correlation of postmortem findings with the presence of heart murmurs enables one to predict fairly accurately the location of the vegetative process. When the



original defect is a congenital lesion the vegetations will be centered about that area involved. In acquired heart disease (almost always rheumatic and only rarely luetic) if there is an apical systolic murmur and no diastolic murmur the vegetations will be confined to the mitral valve. Occasionally there is only a systolic murmur but one that is best heard at the base of the heart due to aortic stenosis without evidence of aortic insufficiency. Under these circumstances there will be vegetations on the aortic valve. When an aortic diastolic murmur is heard there will always be found an active bacterial endocarditis of this valve either alone or with extensions on the mitral valve. When there are typical signs of mitral stenosis without evidence of aortic insufficiency the growths will be found on the mitral valve, but when an aortic diastolic murmur is also present it will be difficult to draw any conclusion for the murmur at the apex may prove to be an Austin Flint phenomenon. In this way one can fairly well predict the location of the vegetative process.

**Differential Diagnosis.**—During the early days this disease offers considerable difficulty in diagnosis. The insidious onset with vague non-localizing symptoms may lead to a diagnosis of grippe. Later as the fever continues and sweats develop tuberculosis will be suspected or because of spots on the skin and enlarged spleen typhoid fever will be considered. More frequently because of the presence of a heart murmur, fever and some vague pains, the condition will be regarded as rheumatic fever. The absence of any murmur is a very reliable clue in eliminating the diagnosis of subacute bacterial endocarditis. I have seen only one instance of this disease in which either a systolic or diastolic murmur was not heard on careful auscultation. Generally the murmur is of more than very slight intensity and in most cases does not change over long periods of time. The sudden development of a new murmur or the accentuation of an old murmur occurs but rarely. This indirect method of ruling out the diagnosis has proved helpful on several occasions. I recall two instances in which the diagnosis of subacute bacterial endocarditis was made by competent physicians on the basis of some of the clinical features discussed above. In the absence of any murmurs I expressed the definite opinion that some other cause would be found for the fever, sweats, etc. In a few days the tubercle bacilli were discovered in the sputum.

The presence of a murmur, on the other hand, does not necessarily mean that there is an active bacterial endocarditis. It obviously is a common finding in rheumatic heart disease and the new infection with fever, sweats and various aches and pains may be a recurrent rheumatic bout with or without rheumatic carditis. This differentiation between rheumatic fever and subacute bacterial endocarditis is sometimes very difficult and always most important. No matter how ill the patient suffering from the former condition may be we are hopeful of a recovery, and no matter how well one with the latter condition may appear we



fear a fatal outcome. Apart from the specific and more characteristic features previously discussed there are some more general differences between the two diseases. The heart rate is apt to be more rapid in relation to the degree of fever in rheumatic than in the bacterial infection. Although both may show a moderate degree of anemia it becomes eventually more marked in the latter condition. Although both show a slight leukocytosis this may be absent in the presence of fever more often in subacute bacterial endocarditis. Salicylates do not alter the fever or the symptoms in the one and generally do so in the other. Pericarditis, arrhythmias and alterations in the electrocardiograms (especially prolonged P-R time) are common in rheumatic and rare in bacterial infections.

The presence of a distinct murmur and its proper interpretation, however, may be the main clue to the diagnosis. I recall an instance when a man while at work was taken with severe pain in the left loin radiating to the genitals. He also noticed grossly bloody urine. It seemed like a definite case of stone in the left kidney or ureter and was so regarded by all the physicians who saw him. There was a moderately loud apical systolic murmur which he had known about for many years. On close questioning I learned that while at work he had not felt really well for several weeks. Although there was no fever at this time, to explain both the murmur and the sudden pain in the kidney, a diagnosis of subacute bacterial endocarditis with left renal embolus was made. This was confirmed by the finding of streptococcus viridans in the blood stream, although the culture was made when the patient's temperature was only 99.2° F. This eventuality is by no means rare in patients who have had a so-called "benign" systolic murmur for many years.

Finally, skin tests may differentiate rheumatic fever from bacterial endocarditis. In 80 to 90 per cent of cases of rheumatic fever, if dead cultures of streptococcus hemolyticus or streptococcus viridans (0.1 to 0.01 c.c.) or the split protein products of these germs are injected intracutaneously, a positive reaction consisting of a definite wheal will result in twenty-four hours. In a fairly large series of cases of subacute bacterial endocarditis such tests have invariably proved entirely negative except in very rare instances. This is a simple test and the reading can be made in twenty-four hours. With our present experiences, if the skin test is positive, one may incline to the opinion that the condition is not a bacterial endocarditis. If it is negative one may suspect this diagnosis but it must be remembered that in about 50 per cent of normal individuals and 10 per cent of rheumatic patients the reaction is also negative. These differences in skin reactions and other suggestive clinical evidences of a certain incompatibility between the rheumatic state and bacterial endocarditis, have led me to think that those individuals who lose their rheumatic predisposition or allergic type of response are the ones who become more susceptible to the development of bacterial endocarditis. The more immune they become to the one, the more



susceptible to the other. It is known that patients who already are suffering from streptococcus viridans endocarditis are highly immune to streptococci. This is evident from the skin test and from the presence of immune bodies in their blood. In fact I have injected living virulent streptococcus in considerable quantities, subcutaneously, in such patients without producing any local reaction or only a very slight one. What is not known is whether this highly immune state precedes or follows the development of the bacterial endocarditis. The simultaneous presence of rheumatic carditis and bacterial endocarditis on postmortem examination in a few cases is not in conflict with the view that there is an antagonism between the two diseases. The bacterial infection, just like any infection, may stir up rheumatic fever and the bacterial infection may have preceded rather than followed the rheumatism. In any event, the skin test has proved of some value in differentiating doubtful cases.

It is generally regarded that emboli and a positive blood culture are the two outstanding and determining evidences of a bacterial endocarditis. This really needs some qualifications. Emboli occur in rheumatic and other types of heart disease in which there is no vegetative endocarditis. In such cases, sterile mural thrombi are generally present in the auricles, from which bits may become dislodged producing peripheral emboli either into the systemic circulation or to the lungs. When such emboli occur, recovery is likely to take place. Likewise, occasionally streptococci may be found in the blood stream by cultural studies when there is no reason to believe they are coming from the valves of the heart. It follows, therefore, that although these points are extremely important the rest of the clinical picture is essential to validate the diagnosis of subacute bacterial endocarditis. Because of the grave prognosis that the diagnosis of subacute bacterial endocarditis entails, most careful consideration must be given to other possibilities like tuberculosis, undulant fever, rheumatic heart disease and the like.

**Prognosis.**—This is one of the most fatal of the common types of cardiovascular disease. Libman has shown both clinically and by pathological studies that recovery and healing can and do take place. This possibility always lends some hope to patients suffering from this disease. Its duration varies considerably from a few months to a year or more. Recoveries, however, have been very rare in the past, if the diagnosis is reserved for those cases that display the complete clinical picture. Very likely in mild cases in which the fever does not rise above 101° F., in which one might hesitate to make a positive diagnosis, spontaneous recovery does take place. On the other hand if a fatal outcome is regarded as essential to the diagnosis there obviously can be no recoveries. There have been instances of satisfactory recovery in which a careful review of the data forces one to accept the diagnosis. It is curious that on very rare occasions a patient may recover symptomatically and yet continue to harbor the streptococci in the blood



stream for a long time. Since the introduction of the sulfa drugs many cures have been reported and now, with the use of penicillin, there is reason to hope that the majority of patients may recover if treatment is started early.

**Treatment.**—A most extensive variety of procedures has been employed in the hope of arresting this infection. Because the disease lasts for months and the general strength of the circulation remains well compensated for a long time, there is ample opportunity to try even extensive and prolonged methods of treatment. A great many different dyes and chemicals have been used intravenously. Sodium cacodylate has been given daily intravenously for weeks at a time. Vaccines and sera have also been given frequent trials. Normal human beings have been immunized by the injection of increasing doses of vaccines of streptococci obtained from the patient's blood stream. This serum was then used therapeutically but without success. The production of sterile abscesses by turpentine injections and even an extensive surgical operation, recommended by Bier, consisting of cauterizing a large part of the under surface of the breast to obtain an aseptic inflammatory reaction, has also been used. I have employed practically all these methods and also induced fever, inoculating the patients with rat-bite fever or malaria but have met only with failure. Transfusions are frequently employed if the red blood count drops below 3.5 million, but it is doubtful whether anything more than a slight amelioration of the symptoms is thereby obtained. Bacteriophage has been administered intravenously and there are isolated cures reported. The same may be said of a special serum obtained from goats. Some cures have been reported by Osgood on a regimen of frequent intravenous injections of neo-salvarsan.

Because of the differences in the allergic state that is present in subacute bacterial endocarditis as contrasted with rheumatic fever I have tried to alter the sensitivity of the patient by the injection of streptococci intradermally. It has seemed that if one could make the patient's skin positive he might then be able to conquer the infection. For this purpose living virulent streptococci were injected into the skin but just as with all other specific methods of treatment this also has proved unsuccessful.

With the discovery of sulfanilamide and its allied chemicals the previous pessimistic and almost hopeless outlook has changed. Numerous cures have been obtained. Whereas formerly I had not seen a single recovery in over one hundred cases of definite subacute bacterial endocarditis, I now have seen four unequivocal cases with positive blood cultures and other clinical findings, in which recovery took place on sulfa therapy. These patients became ambulatory and symptom free. Although this method of treatment is being supplanted by penicillin, it might be of interest to outline its general form.

In the first place the physician must realize that the problem is difficult and involved and will not be solved at home. The patient must,



therefore, be hospitalized. Once the diagnosis has been made, and this should be done as rapidly as possible, sulfa therapy is started. Sulfadiazine appears to be the drug of choice, although in vitro experiments should be carried out to see which is most effective for the strain of streptococcus grown in the particular case. Nine grams are given the first day in divided doses and 1.0 to 1.5 grams six times daily at four-hour intervals thereafter. The blood level should be kept at about 10 milligrams. With this, an equal amount of sodium bicarbonate is given and all the other procedures carried on as indicated. It is hoped to bring the temperature to normal or at least to a lower level. After several days, fever therapy is started while continuing sulfadiazine. It is thought that artificial fever enhances the effect of sulfa drugs, though bacteriological tests do not necessarily confirm this. Fever may be administered in a "hot box" or by the intravenous injections of typhoid vaccine. The treatments are given every other day and it is desired to maintain a temperature of 104° F. or more for at least four hours. When using typhoid bacilli the first dose is 25 million organisms. This is increased with each dose by 50 to 100 per cent, varying the amount according to the reactions obtained. A series of about twelve fever treatments are given. More satisfactory effects occasionally may be obtained if the vaccine is given slowly in a saline infusion and organisms added if the temperature falls too soon. Sulfadiazine should be continued with decreasing doses after this for some months even if the temperature remains normal and the blood cultures sterile.

What appears to be the most effective treatment for patients with acute or subacute bacterial endocarditis is penicillin. It is too early to formulate a final appraisal of the results, but they seem to be amazing. When the drug was first used it was uniformly disappointing because, as was found out later, the dose was too small. Loewe and his collaborators then reported a group of cures obtained through the use of huge doses. Their procedure is now the one generally employed. The diagnosis should be made as quickly as possible, for in early cases less of the drug and a shorter course of treatment will be required and response will be obtained more readily. The sensitivity of the organism cultured from the blood stream should be tested in vitro to various concentrations of penicillin. It is then possible to calculate whether or not an adequate concentration of the drug is obtained in the blood stream of the patient when it is administered therapeutically.

Penicillin is best given by continuous intravenous drip using about 1200 to 2500 c.c. of 5 per cent glucose solution. The number of units will range between 200,000 to 400,000 daily. As the supply of the drug becomes greater there is reason to believe that even larger doses might be desirable. There appears to be no toxic reactions even from excessive doses. It is best to avoid a saline solution as the patients have organic heart disease and may develop pulmonary edema and congestive heart failure following the use of the salt solution. In fact, large amounts of



liquid administered intravenously are also undesirable. However, if the intravenous drip is too slow (15 to 20 drops a minute) as would be necessary when only 1200 c.c. are given in twenty-four hours, the veins may thrombose or the needle may clog. The physician should be ready to change the site of injection from one vein to another if the flow stops. At times intramuscular injections may be employed, especially if veins are not available. Then injections should be made every two to three hours all day long. A course of treatment should be not less than two weeks and may need to be continued for three weeks or longer.

As penicillin treatment is started it is amazing that within forty-eight hours the temperature may become normal and the patient may quickly feel marked symptomatic relief. Occasionally unexpected rises in temperature may appear that possibly result from local thromboses of the veins that are being used or from the intravenous apparatus (tubing, etc.).

It has been claimed that the simultaneous administration of heparin intravenously or intramuscularly during the course of penicillin treatment will obtain better results. This has not been convincingly proved as yet. Already I have observed several patients who have been cured by penicillin without heparin or sulfa drugs. How long these patients will remain well, only time will tell. It is fair to say that if they continue fever free, with negative cultures, and feel well for six months or longer, as some of them have already done, and later develop the disease again, a new infection probably will have occurred. After all, the valves remain deformed and continue to be a focus upon which bacteria may lodge.

All in all this new chemotherapy promises to be a great advance in the treatment of a disease that previously was almost invariably fatal, just as it already has proved to be in many other serious infectious diseases. Whether or not the simultaneous use of heparin or sulfadiazine will be more effective than penicillin alone, will be known in the near future.

The difficulty with all the clinical methods of treatment is that, even if the blood stream becomes sterilized, bacteria may continue to grow inside the vegetations that persist on the valves. The bactericidal agents cannot penetrate the fibrin and tissue that surround the causative organisms. For this reason attempts have been made to dissolve the vegetations by the constant intravenous injection of heparin while the patient is receiving sulfa therapy. Although rare cures have been reported following this technic, I have had no recoveries and have felt that heparin was responsible for cerebral accidents in some of the cases. Likewise, dicoumarin given orally, which has an effect somewhat similar to heparin, has been used but the ultimate value of this has not been established.

**Prevention.**—Now that we have drugs that are so effective against many infections it is imperative to use them in preventing this devastating disease whenever possible. Certainly, whenever a patient having rheu-



matic or congenital heart disease is to have a tooth extracted or a tonsillectomy or any operation that might entail the possibility of infection, sulfadiazine or a similar drug should be given for two days before and after the operation. About 3 to 4 grams daily would seem to be sufficient for this purpose. The question comes up whether or not it would be advisable to give vulnerable individuals small doses (1.5 to 2.0 grams) daily for the rest of their lives. This might well apply to cases in which the statistical incidence of bacterial endocarditis is between 25 and 40 per cent, *i.e.*, interventricular septal defect, patent ductus, and well compensated rheumatic aortic or mitral insufficiency with regular rhythm.



## 11

### CONGENITAL HEART DISEASE

**General Considerations.**—Congenital defects of the heart are by no means rare in infants but because many such abnormalities are not compatible with a normal development, the incidence in the general population with increasing years diminishes very rapidly. Probably not more than 1 or 2 per cent of all those having organic heart disease over the age of twelve years are suffering from congenital abnormalities. This figure will naturally be much greater for children under twelve years of age. There is very little known as to the fundamental cause of these defects except that there is a distinct hereditary factor.

During the first days, weeks or months of life the diagnosis of the exact type of defect is extremely difficult and generally impossible to make. The abnormalities of the heart are much more often multiple than single and the physical signs are not sufficiently distinctive. As months and years go on and the child survives, the clinical picture becomes more definite so that in older children combinations of physical findings occur that lend themselves to more accurate analysis. The result is that in those over twelve years of age it is often possible to make a correct diagnosis of at least the major defect, if there is more than one, and not infrequently of all the congenital abnormalities.

Inasmuch as some of the evidence of congenital heart disease may be apparent at birth or shortly afterward, physicians should always note the presence or absence of heart murmurs or cyanosis when a child is born. Such examinations should be repeated a few weeks and a few months after birth as the signs may not be detectable immediately after birth. It is more difficult but also important to elicit evidence of cardiac enlargement during the first few months of life. If proper



notations are made at this time much confusion that often arises in later life, as to whether a certain murmur developed after an infection or was there from birth, will disappear.

It is not the purpose of this discussion to take up the numerous rarities of congenital cardiac abnormalities. Attention will be focused on several conditions that are met with in older children and in adults which are common enough and sufficiently discrete to merit emphasis.

It is well known that cyanosis is often a striking feature in congenital heart disease. This cyanosis has a different mechanism than that which is seen in acquired heart disease. In the former the cyanosis is due to venous blood reaching the arterial blood in the chambers of the left heart or aorta without going through the lungs. It indicates a right to left (venous-arterial) shunt through one of the septa of the heart or great vessels. If there is a defect in the auricular or ventricular septum, some of the venous blood may pour into the left auricle or ventricle from the right chambers and become admixed with the arterial blood. This then is expelled through the aorta to the general circulation and if there is a sufficient amount of unaerated venous blood, cyanosis will be present. Whether cyanosis does develop in cases of a septal defect will depend on the size of the aperture and the pressure relations in the two sides of the heart. Inasmuch as the systolic pressure in the left ventricle is greater than in the right, the flow of blood through a ventricular septal defect will be from left to right and there need be no cyanosis. If, however, the septal defect is great there is an appreciable flow of blood from right to left during the diastolic filling of the ventricle when the intraventricular pressure is nil or very slight. Furthermore, if for any reason the pressure in the right side becomes greater, as during violent coughing or the pressure in the left side falls as in left ventricular failure, the balance may favor a flow of blood from the right to the left and one may see cyanosis develop. Finally venous blood may be expelled into the peripheral circulation because the aorta comes off completely or partly from the right ventricle. Therefore, it must be clear that cyanosis in congenital heart disease does not arise because of congestion in the lungs with a slow pulmonary circulation and the resultant insufficient oxygenation of the blood as occurs to some extent in ordinary congestive heart failure, or because of peripheral venous stasis, but is due to the failure of some of the venous blood ever to reach the lungs. When heart failure develops in congenital cardiacs both mechanisms may be at work.

It is of some interest to note at this point that cyanosis has been confused with argyria. I have seen a considerable number of instances in which patients were treated for heart disease because of prolonged "cyanosis" and in which the discoloration of the skin was the result of long-continued use of silver-containing nose drops. One was a boy nine years old who had been regarded as a congenital cardiac since infancy. The mother had instilled nose drops once or twice a day since



the age of three months because of a "rhinitis and sinusitis." The skin had the typical slate-like discoloration of argyria. The differential diagnosis was quite simple. Apart from the fact that the heart was not enlarged and showed no murmurs, the tongue, the mucous membranes of the mouth and the conjunctivae were all normal in appearance. In a congenital cardiac with such a degree of discoloration of the skin these tissues would also have shown marked cyanosis. Another case was that of an adult who had also used nose drops for many years and because of the supposed cyanosis was given digitalis and treated for some bizarre form of heart disease.

Another confusion that may rarely arise is between methemoglobinemia and congenital heart disease. I recently saw a young man in the early twenties who had been markedly cyanosed since birth. Although he had had fair health he gradually developed weakness and dyspnea on effort, especially on climbing stairs. Physical examination including that of the heart revealed no abnormalities. Cyanosis was very striking and of the type typically seen in classical tetralogy of Fallot. The absence of clubbing, of cardiac murmurs or abnormalities demonstrated in electrocardiograms made the case very puzzling. The resident physician at the Peter Bent Brigham Hospital, Dr. C. B. Favour, suspecting the proper diagnosis, bubbled oxygen through some venous blood withdrawn from the arm. It was found that instead of turning red the blood retained the blue color. This pointed to the diagnosis of methemoglobinemia which was confirmed by chemical and spectroscopic tests. Within one hour after the intravenous injection of 1.0 milligram of methylene blue per kilogram of body weight the color of the skin was normal. The patient's symptoms all quickly disappeared. This extraordinary cure was subsequently maintained by administering about a 0.2 gram capsule of methylene blue by mouth daily.

The second striking feature is clubbing of the fingers and toes. This accompanies the prolonged cyanosis and does not come merely because there is a congenital defect. One naturally ascribes the clubbing to the anoxemia in the arterial blood attendant to the cyanosis. The exact relationship, however, is not clear for clubbing of the fingers is found in a variety of conditions, in many of which there is no cyanosis or anoxemia. It occurs in subacute bacterial endocarditis when there is no heart failure whatever. It is common in abscess of the lung when cyanosis and anoxemia are absent. It does occur in other conditions with prolonged cyanosis such as chronic emphysema. If there is a single cause for the clubbing, it has not as yet been discovered.

Furthermore, congenital heart disease is often, but by no means invariably, productive of loud murmurs. When such murmurs are heard in infants or children, unless there is some other obvious cause, such as anemia or an active rheumatic infection, a congenital defect must be suspected. Pulmonary tuberculosis is a not infrequent development in those suffering from congenital heart disease. It is of some



interest that acquired rheumatic heart disease is fairly common amongst congenital cardiacs who reach adult life. Of greater importance is the fact that bacterial endocarditis is a frequent complication and cause of death in many persons with congenital abnormalities. The bacteria have a predilection for any abnormality of the internal lining of the heart or great vessels. Stunted or retarded bodily growth may also result from congenital heart disease when it is accompanied by an appreciably diminished outflow of blood through the aorta. Finally it is commonly associated with other congenital abnormalities and with mental retardation or idiocy.

**Idiopathic Hypertrophy of the Heart.**—Occasionally idiopathic hypertrophy of the heart may be congenital. Such patients merely show cardiac enlargement without any valvular or septal defects. They rarely live longer than a few years and present the picture of increasing cardiac embarrassment with dyspnea. The cause of the enlargement is not known but one might suspect that a careful study of the coronary circulation would reveal some defect either in the larger or the very fine branches in some of these cases. In a few cases considered as congenital cardiac hypertrophy there is reason to believe that the condition was due to faulty metabolism, because extensive glycogen deposits were found in the heart muscle. Others probably were the result of prolonged anemia or avitaminosis and possibly to paroxysmal rapid heart action, all of which can cause cardiac dilatation. Despite this there remains a few in which no cause can be found.

**Coarctation of the Aorta.**—A much more common congenital anomaly is coarctation of the aorta. Although there are different degrees and locations for this narrowing the most common site is at the arch of the aorta just beyond the left subclavian artery at the point where the ductus arteriosus develops. This ductus often remains patent in cases of coarctation and frequently the foramen ovale is also open. The constriction of the aorta leads to a definite sequence of findings, more or less extensive, depending on the degree of narrowing. The ascending aorta proximal to the coarctation becomes dilated and although there may be a slight dilation of the aorta for a short way beyond the constriction, for the most part the thoracic and abdominal aorta is much smaller than normal. Extensive and enormous anastomotic arteries develop which bridge the blood stream from the first portion of the aorta proximal to the constriction to the lower part of the body. The internal mammary, the intercostal and other arteries increase tremendously in size.

As a result of these abnormalities definite objective findings become manifest that enable one to make a satisfactory clinical diagnosis. Not so long ago all cases of coarctation of the aorta were first detected on postmortem examination, then except on very rare occasions the roentgenologist would first make the diagnosis, but now it often can be made by the clinician. Adults with this condition develop hyper-



tension in the arms although the blood pressure in the legs is lower than normal. Normally the pressure in the legs should be higher than in the arms. Inasmuch as determinations are not made routinely of the blood pressure in the legs, this discrepancy can easily be overlooked. It should become the habit of all physicians to feel for pulsations over the abdominal aorta and femoral arteries and if they are absent or markedly diminished, especially if the patient has hypertension and if there is a basal systolic murmur, coarctation of the aorta should be suspected and measurements of the blood pressure in the legs should be made. If the pressure in the legs is less than in the arms, a presumptive diagnosis can be made. A second feature is the finding of pulsating superficial arteries over the back, around the scapulae. Wormlike pulsating subcutaneous arteries may be found there. Examination of the heart may show some enlargement and murmurs at the base of the heart of the type seen in hypertension, aortic valvular disease or patent ductus arteriosus, *i.e.*, there may be systolic with or without diastolic murmur. The systolic murmur which is always heard at the base of the heart may be as loud or louder in the interscapular region. Finally the *x*-ray of the chest generally can establish a conclusive diagnosis. This will show a prominent ascending aorta and an absence of the aortic knob of the descending aorta. It will also show a peculiar notching or scalloping of the lower margins of the ribs due to the erosion produced by the enlarged pulsating intercostal arteries. These changes are pathognomonic of coarctation of the aorta.

When it is appreciated that about 0.05 per cent of the entire population have this abnormality it becomes important for the physician to become familiar with it. These patients often survive to adult life and may even reach old age. Their difficulties are those that accompany vascular hypertension, *i.e.*, myocardial failure and cerebral hemorrhage. Besides this they are prone to two other peculiar complications, *i.e.*, rupture of the aorta and bacterial endocarditis. As to treatment they should be advised along the lines customarily given to a patient who is subject to the above disasters, bearing in mind that activities often need not be restricted a great deal.

**Dextrocardia.**—Congenital dextrocardia is of two types. In the first the apex of the heart points to the right but is formed by the right ventricle. The heart is essentially rotated more to the right so that the left ventricle lies more anterior than normally but yet to the left of the right ventricle. This condition is due to a congenital arrest in development and therefore is generally associated with other cardiac abnormalities such as absence or defects of the septa, transposition of the arterial trunks, etc. Such patients are apt to suffer serious handicaps. The second type is called the "mirror-picture" dextrocardia and is practically always associated with a complete transposition of the other viscera (the liver and appendix being on the left, the spleen on the right, etc.). Here the apex of the heart points to the right, is felt near the right



nipple but is made up of the "left ventricle" (the one receiving blood from the lungs). The right ventricle and auricle lie to the left. There are no other defects in the heart and such patients have no symptoms referable to the circulation. This condition is often overlooked until accidentally found on routine examination. It cannot be regarded as an arrest in the development. It suggests rather an inversion of the embryo in its relation to its primitive yolk sac. The electrocardiograms in mirror-picture dextrocardia are absolutely pathognomonic. Lead I will show inverted P and R waves just as would result if the lead wires in taking a normal Lead I were accidentally reversed (Chapter 21, Fig. 97). Patients with this condition have a normal life expectancy and need not be restricted in their activities.

**Patent Foramen Ovale and Atrial Septal Defect.**—Patency of the foramen ovale is probably the most common of all congenital cardiac anomalies. In intra-uterine life this opening in the interauricular septum permits the blood to flow from the right to the left side of the heart without going to the lungs, for the latter are compressed and functionless. During the first few months the foramen ovale normally closes but in a considerable number of individuals a greater or lesser patency persists. When the foramen ovale is small it produces neither signs nor symptoms of heart disease. When it is large it may result in enlargement and dilatation of the right auricle and ventricle and the pulmonary artery. There may or may not be any murmurs and congestive heart failure due to strain on the right side of the heart may result. Paradoxical embolism may occur in this condition as in any instance in which defects in the septa of the heart are present. Under such circumstances a thrombus from a peripheral vein or from the right auricle on becoming dislodged may go through the septal defect and produce an arterial embolus (to brain, kidney, spleen, etc.).

There are other defects of the interauricular septum which are similar in many respects to patency of the foramen ovale. The lower part of the septum (*persistent ostium primum*) or the upper portion (*persistent ostium secundum*) may be patent. The entire auricular septum may be absent producing a "cor triloculare biventriculare." All these auricular defects produce similar changes, the degree of which depends to some extent on the size of the patency. The right auricle and ventricle may become enormously dilated and hypertrophied and the pulmonary artery considerably enlarged while the aorta is actually smaller than normal. Slight cyanosis is common and clubbing is rare. These cases of atrial septal defect may show no murmurs, or there may be a slight basal systolic and occasionally a pulmonary diastolic murmur. Right axis deviation in the electrocardiograms is common. I have seen two instances in which the pulmonary artery was so dilated that it compressed the left recurrent laryngeal nerve causing paralysis of the left vocal cord. Some patients with interauricular septal defect also have mitral stenosis (Lutembacher syndrome) and they then may also have auricular



fibrillation. In general patients with atrial septal defect almost never develop bacterial endocarditis and may live fairly long and useful lives.

**Patent Ventricular Septum.**—Interventricular septal defects are quite common and rank next in frequency to auricular defects. They are generally associated with other abnormalities such as right-sided position of the aorta or pulmonary stenosis but may occur as the only lesion (Roger's disease). The patency is usually small, measuring 1 to 2 centimeters in diameter and is most often located in the upper part of the septum just below the aortic valve. This condition generally produces no symptoms of cardiac embarrassment, but despite this very few live beyond the age of thirty or forty. Probably the cause of this is that subacute bacterial endocarditis occurs in about 40 per cent of the cases. When bacterial endocarditis does occur the vegetations are apt to be located in a circular fashion around the right ventricular aspect of the defect or on the right ventricular wall opposite the defect. A rapid jet of blood is propelled from the left to right ventricle and bombards this portion of right ventricular wall producing a localized fibrous or even calcified area. The heart may be slightly enlarged and merely show a harsh systolic murmur, best heard over the lower left portion of the sternum. A systolic thrill is also often present. The location of the defect near the bundle of His accounts for the frequent association of congenital heart block or defective intraventricular conduction with this abnormality. The electrocardiograms may show biphasic initial ventricular complexes. There is no clubbing or cyanosis, unless the aorta partially overrides the right ventricle.

**Tetralogy of Fallot.**—Of much more importance, because of its greater frequency, is a combination of ventricular septal defect, pulmonary stenosis, right ventricular enlargement and dextraposition of the aorta ("tetralogy of Fallot"). This is the most common cause of marked congenital cyanosis met with in adults. Although most of these patients do not survive to adult life, some carry on very well to the fifties or more. With this condition there is an appreciable right to left shunt because venous blood is being partly expelled through the ventricular defect into the left ventricle and because venous blood from the right ventricle is delivered directly into the aorta which overrides it. The result of the prolonged cyanosis is marked clubbing of the fingers and a compensatory polycythemia, the red blood count often reaching 10 million or more. Direct examination of the heart will show right ventricular enlargement with marked preponderant hypertrophy of the right ventricle in the electrocardiograms. When these electrocardiographic changes are very striking one can be sure that congenital heart disease probably with pulmonary stenosis with or without other defects is present. A loud systolic murmur is present all over the precordium, best heard at the pulmonary area and generally accompanied by a systolic thrill in this region. The x-ray may show a diminished promi-



nence and concavity of the pulmonary artery and a projection to the right of the aorta.

**Eisenmenger Complex.**—The Eisenmenger complex is a much rarer complex that resembles the tetralogy of Fallot. It consists of a ventricular septal defect, dextraposition of the aorta, but no stenosis of the pulmonary valve although there may be pulmonary insufficiency. Cyanosis is slight or moderate and clubbing may be slight or absent. A moderately loud systolic murmur will be present over the mid-precordium and possibly a pulmonary diastolic murmur. The electrocardiogram will show right axis deviation.

**Pulmonary Stenosis.**—Pulmonary stenosis as an isolated lesion is quite rare. The evidence of this anomaly is the presence of a rather loud systolic murmur in the second left interspace transmitted to the left back and a systolic thrill in the pulmonary area. There is also marked right ventricular hypertrophy in the electrocardiograms as a result of the increased work of the right side of the heart.

**Patent Ductus Arteriosus (Botalli).**—During fetal life blood leaving the right ventricle is expelled through the pulmonary artery, then by way of the ductus arteriosus into the aorta and in this way to the placenta where it becomes oxygenated. The blood obviously does not go to the lungs, which are compressed and atelectatic. During the first week of life the ductus arteriosus having lost its function, when the venous blood begins to flow to the lungs, normally disappears. When this opening between the pulmonary artery and the aorta fails to close the condition called patent ductus Botalli results. Although this is commonly accompanied by other congenital abnormalities it often is present as the sole or the major defect. This does not produce cyanosis or clubbing because the flow of blood is from aorta to pulmonary artery (arterial to venous) and there results no admixture of unoxygenated blood in the systemic circulation.

There need be no symptoms of circulatory distress from patent ductus arteriosus and its presence is often detected accidentally on routine examination. The systolic blood pressure is normal but the diastolic is low just as occurs in aortic insufficiency or any arteriovenous fistula. The main diagnostic finding is a peculiar murmur heard best in the second left intercostal space and often just to the right of the left mid-scapular region. The murmur when typical is continuous throughout systole and diastole and has been called a machinery murmur because of its quality. It may rarely last only through systole and is often accompanied by a systolic thrill in the pulmonary area. The typical murmur seems to envelop the pulmonary second sound and is loudest during the latter part of systole (Fig. 156). *x*-Ray examination may also aid in the diagnosis in showing a marked prominence of the pulmonary artery which becomes dilated. Other roentgenological findings in the order of frequency are: slight cardiac enlargement, slight dilatation of the left auricle, engorged pulmonary vessels and exag-



gerated pulsations of the left ventricle and pulmonary artery. The electrocardiogram either shows normal tracings or may indicate slight left ventricular preponderance. The condition is compatible with good health and is often seen in young adults. In some, however, bodily development is stunted because of the diminished outflow of blood through the aorta and there may also be weakness and dyspnea on effort. Pulmonary tuberculosis is not infrequent and bacterial endocarditis is a quite common complication of this defect and probably explains why it is extremely rare in older individuals.

In 1938 the first successful ligation of a patent ductus arteriosus was reported by Gross and Hubbard. Since then many patients with this anomaly have similarly been operated upon. The operation seems to be simple and effective. I have seen many of these patients and feel confident that in properly selected cases this abnormality can be adequately treated by surgical means. The loud murmurs promptly disappear and the low diastolic pressure due to the shunt of blood from aorta to pulmonary artery immediately returns to normal. The purposes of the operation are to prevent the subsequent development of bacterial endocarditis which has been a complication in about 25 to 40 per cent of the cases of patent ductus arteriosus, to prevent the retardation of mental and physical development and to improve the efficiency of the circulation when there is any evidence of cardiac insufficiency. The latter two objectives are obtained by stopping the considerable loss of arterial blood that leaks back into the pulmonary circuit. A successful operation also will diminish the work of the overburdened left ventricle and thereby delay or prevent heart failure. Needless to say, in the selection of cases the diagnosis must be certain and, for the present, subjects having other congenital abnormalities should not be considered.

In experienced hands, the operative mortality at present is about 5 to 10 per cent and very likely will be further reduced before long. Inasmuch as the average age at death of a large group of patients with patent ductus arteriosus has been found to be only thirty-five years, successful operations ought to lengthen life expectancy considerably.

It is of particular interest that patients with subacute bacterial endocarditis engrafted on patent ductus can be cured by this operation. It is advisable to give one of the sulfa drugs, preferably sulfadiazine, or penicillin in adequate doses before and after the operation, although such patients have been cured without chemotherapy. It is amazing that blood cultures that have been repeatedly positive up to the moment of operation can become negative within minutes after the division of the duct. In some, cultures continue positive for some days or even weeks post-operatively and yet cures result. It is most important to institute treatment early, for if the vegetations have spread well beyond the duct, especially to the aortic or mitral valve, recovery will not be obtained. The occurrence of arterial emboli will indicate that the vegetative process has extended too far and will spell a fatal outcome. Splenic



enlargement or hematuria are not necessarily contraindications for operation.

**Right Aortic Arch (Dysphagia Lusoria).**—There is one congenital abnormality that is of some importance because of the peculiar associated clinical findings, *i.e.*, right aortic arch. Very rarely the arch of the aorta is directed to the right and is formed by the primitive fourth right aortic arch. The aorta then passes over the right bronchus to the right and behind the esophagus and trachea. The pressure on the esophagus or trachea may cause difficulty in swallowing ("dysphagia lusoria") or respiratory symptoms like cough and wheezing dyspnea. Esophagoscopy may then reveal a constriction and the *x*-ray examination following a barium meal will show that the arch of the aorta turns to the right and lies behind the esophagus compressing it forward and to the left. In addition there may be remnants of the congenital cords which formed the left aortic arches, thereby forming a ring around the esophagus and trachea. This anatomical relationship is of some importance because it is hoped that a surgical attempt to divide such constricting cords may some day be curative. So far therapy has been limited to advising a soft or liquid diet when dysphagia develops.

**Bicuspid Valves.**—Congenital bicuspid aortic or pulmonary valves are clinically of importance only in so far as they are the frequent site upon which acute or subacute endocarditis develops. Such lesions may account for some of the instances in which bacterial endocarditis develops in patients who previously showed no murmurs in the heart. When bacterial endocarditis develops in patients who have not suffered previously from a rheumatic infection careful search of bicuspid semi-lunar valves should be made.

Although there are numerous other congenital cardiac abnormalities either occurring singly or in varied combinations they are not sufficiently important to deserve discussion in this review.

**Treatment for Congenital Heart Disease.**—There is no known treatment for congenital heart disease, except the operation for patent ductus arteriosus mentioned above. Digitalis is generally useless but quinidine may be helpful for cardiac irregularities present in some cases. Inasmuch as pulmonary tuberculosis and bacterial endocarditis are common complications, efforts should be directed at maintaining as high a general bodily resistance to infection as possible. Phlebotomy may be helpful when there is marked cyanosis and plethora and especially if there are cerebral symptoms. Patients should be encouraged to carry on some moderate occupation whenever possible, since many are able to live a fairly long, useful life.



## FUNCTIONAL HEART DISEASE

FOR practical purposes it seems convenient and helpful to classify all patients having abnormal signs or symptoms of heart disease in whom there is no structural disease of the heart as suffering from functional heart disease. This will naturally include a great variety of conditions. In general there will be two groups of such patients, those having some abnormal physical findings in the heart and others with symptoms but without abnormal findings. There will be many, of course, who will manifest both symptoms and signs. There are numerous terms in use to designate these conditions and although they may connote slightly different states, they all may properly be used to specify functional heart disease in the sense described above. Among the current terms are "neurocirculatory asthenia," "effort syndrome," "disorderly action of the heart," "soldier's heart," "cardiac neurosis" and "nervous heart." It will be seen in the succeeding paragraphs that other designations may well be used in this same group of patients such as "benign cardiac irregularity" and "benign systolic murmur."

Let us first consider the group of patients who have either no symptoms whatever or insignificant ones, who show abnormal signs. During the course of an insurance examination or any other routine examination the physician may detect a faint systolic murmur in the absence of any other evidence of heart disease. This signifies the absence of hypertension, cardiac hypertrophy, diastolic murmurs, or any other cardiac symptoms. If there is no history of any previous rheumatic infection, under these circumstances, a slight systolic murmur can be disregarded and called functional or benign. Such a patient, for purposes of classification, may be said to have functional heart disease (benign systolic murmur). This applies only to a faint systolic murmur.

It will be found useful to estimate the intensity of systolic murmurs just as we do the amount of albumin in the urine. It is also necessary to confine the term "systolic murmur" to a bruit that has an appreciable duration and lasts for a definite interval after the first heart sound. Many mistaken diagnoses are made in designating as murmurs a prolonged first heart sound, frequently detected in hyperactive hearts and thin-chested individuals, when the interval between the first and second heart sounds is entirely clear. A more detailed discussion of the systolic murmur will be found in Chapter 17. Suffice it at this point to bear in mind that one should hesitate in considering loud murmurs as insignificant, benign or functional.

2. Apart from the heart itself, the more important factors that seem to be responsible for systolic murmurs are anemia, hyperthyroidism,



## FUNCTIONAL HEART DISEASE

hypertension, fever, tachycardia and the emotional state of the patient. In general it has been found that faint systolic murmurs, although present in only a small percentage of people, should be regarded as having no significance. Those of moderate intensity may indicate some organic disease in the heart or elsewhere but also occasionally occur as a functional manifestation, and murmurs of louder intensity practically always signify some obvious organic disease.

In an extensive study of systolic murmurs it did not seem to matter whether faint systolic murmurs were heard at the apex or base of the heart or whether the patient was examined in the upright or recumbent position, although the frequency of benign systolic murmurs varied somewhat under these different circumstances. It is of no importance whatever that a systolic murmur, previously not present, is brought out by effort, for it was found that in practically all normal individuals systolic murmurs appeared directly after a brief brisk effort. The important point in this regard is that systolic murmurs of greater than the faintest intensity deserve investigation. This simple finding may be an important clue to the diagnosis of otherwise unsuspected hyperthyroidism or bacterial endocarditis. Although in some cases no adequate explanation will be found for the presence of a systolic murmur and such an abnormality is consistent with a long and active life, there is a tendency at present to pay too little attention to systolic murmurs and consider them all as benign. Apart from the conditions just mentioned, this type of "benign" systolic murmur often proves to be a manifestation of rheumatic heart disease, frequently is found to be eventually associated with stenosis of the aortic or mitral valve, occasionally is an evidence of myocardial disease and rarely of congenital heart disease. Notwithstanding these considerations there remain some individuals with faint systolic murmurs who must at present be regarded as having no organic disease and in whom the diagnosis of functional heart disease (benign systolic murmur) must be made.

Further physical findings frequently regarded as benign or functional are certain arrhythmias. Almost all the arrhythmias that are met with in general practice can at times occur in patients who have no organic heart disease. The presence of sinus arrhythmia and extrasystoles of any type is frequently the cause of apprehension and sometimes of discomfort when the condition is entirely functional. The same is true of paroxysmal auricular tachycardia. As our experience has broadened we have come to realize that even auricular fibrillation and flutter, paroxysmal or permanent, may occasionally be unassociated with any other disease. On rare occasions ventricular tachycardia has been observed in otherwise normal individuals. All these irregularities of the heart of course do occur with organic disease as well and it becomes very important to use every reasonable means to exclude organic conditions before making the diagnosis of functional heart disease (benign irregularity). It is particularly important not to overlook hyperthyroidism when auricular fibril-



lation is present as this may be the sole manifestation of a toxic goiter and it will be necessary to make intelligent interpretation of the basal metabolism.

If any of the above irregularities are present without symptoms of angina or congestive heart failure and if other features like hypertrophy of the heart, hypertension, a diastolic murmur, significant changes in the electrocardiogram, a past history of rheumatic infection, abnormal configuration of the x-ray shadow of the heart and aorta or a positive Wassermann reaction are absent, the condition must be regarded as functional. It does not follow that all these investigations are necessary to arrive at this conclusion, but these are the various points to be considered in the study of such cases in which the primary complaint is apt to be palpitation of the heart.

The sensations that develop from benign extrasystoles are so peculiar and at times so distinctive that one should be familiar with them, as often the description of the symptoms given by the patient establishes the diagnosis even without any further examination. They are frequently described as a "flop of the heart," "the heart turns a somersault," "it skips or hesitates," there is "a sudden thump or jump or choking sensation in the throat," "the heart suddenly sinks or a wave passes over me." Although the actual terms used are varied they are all characteristically descriptive of what is going on. Some compare the sensation to "the sudden flapping of a bird's wings" or to a "fish suddenly turning in the water." These disturbances are most apt to occur when the patient is at rest especially while quietly seated or while trying to fall asleep. This is explained either by the fact that the mind is not occupied with other things or, more likely, on the basis that while the subject is at rest the heart rate is slower and the opportunity is greater during the longer diastolic pauses for premature beats to arise. At any rate the sensations are generally absent while the patient is active, walking or busily engaged in his affairs, although there is one rarer type that is brought on particularly by effort.

Although it has long been known that extrasystoles frequently are of no serious significance and that perfectly well individuals may have them, only recently has it been possible to ascribe a definite neurogenic origin to them. Beattie and Brow made animal experiments reproducing persistent ventricular extrasystoles and found that if certain nerve tracts coming from the hypothalamic region were cut the irregularity disappeared. It is of some interest that the hypothalamic region is concerned with the control of our emotions. They also found that if these tracts were cut beforehand the extrasystoles could not be produced by the same technique that always brought them out in animals not subjected to this treatment. In other words, there is a center in the brain that can initiate or that is intimately connected with the formation of premature heart beats. This work has firmly established a structural neurogenic basis for conditions that have long been regarded as functional or nervous.



Treatment for patients with extrasystoles varies considerably. When the extrasystoles come infrequently all that is needed is an explanation that nothing serious is going on. The patient should be encouraged to carry on normal physical duties. It will only rarely be necessary to inhibit the use of coffee or tobacco. When the irregularity is very disturbing, quinidine sulfate in doses of 0.2 to 0.3 grams two or three times daily, or even less, is often effective. Recently papaverine (0.1 gram) administered three times a day by mouth has been advised. At times the patient learns that a certain medication is effective but only necessary just before the palpitation is expected to occur.

It has been stated that auricular fibrillation which generally is associated with organic heart disease can be at times a purely functional disturbance. This is particularly true of the paroxysmal type of auricular fibrillation, but I believe it also occurs in a few cases when the arrhythmia is permanent. In the absence of rheumatic heart disease, especially mitral stenosis, of coronary artery or hypertensive heart disease, one must always suspect that there is an underlying hyperthyroidism when auricular fibrillation, either transient or permanent, is present. If the basal metabolic rate is normal the possibility of a toxic thyroid gland as the cause of the auricular fibrillation is ruled out for the most part. This is not invariably true for it is now believed that even some of these patients with a normal basal metabolic rate already have disease of the thyroid gland and will subsequently show an elevated rate and that the auricular fibrillation may disappear after subtotal thyroidectomy. It has seemed that some such patients have been improved without surgery by taking Lugol's solution. It is also interesting that most of these apparently normal patients whom I have seen with auricular fibrillation and a normal metabolic rate have looked alike, have been males and resemble in some ways patients with active hyperthyroidism.

When hyperthyroidism alone is the cause of the auricular fibrillation, the irregularity may still be regarded as functional for it disappears under appropriate treatment of the thyroid gland and there remains no evidence of organic heart disease. Some work has been done showing that in hyperthyroid animals transient auricular fibrillation can be reproduced by the injection of adrenalin. It was found that without adrenalin such animals would never show auricular fibrillation and that adrenalin in normal animals produced only ventricular irregularities and not auricular fibrillation. One might infer that in patients with hyperthyroidism the secretion from the adrenals has some relation to the production of auricular fibrillation. This would explain the occurrence of transient spells of this irregularity following emotional shocks and fright. At any rate it has become clear that permanent structural disease of the heart is not a necessary prerequisite for auricular fibrillation.

The other general type of patients comprises those who have various symptoms in whom abnormal physical signs like irregularities may or may not be present. Such patients are often young and they complain



of palpitation, weakness, giddiness, pain in the region of the heart and frequently shortness of breath. The palpitation generally is associated with a normal heart rhythm. They merely feel the pounding of the heart which has either a normal rate or is slightly accelerated. When the rate is found to be rapid while the patient is awake it will be normal during sleep. The pain is almost always apical rather than sternal and may be of a dull, constant aching character or momentary and stabbing. When there is shortness of breath it may be merely of the type that accompanies a state of fatigue as if it were a greater burden to lift the chest or it is due to a peculiar "sighing type" of breathing. This latter phenomenon is sufficiently common and characteristic to require special emphasis. We frequently see patients, often young women, who complain of shortness of breath without any other evidence of heart disease. On questioning them it will be found that the dyspnea occurs particularly at rest. It is obvious that true cardiac dyspnea at rest indicates a very grave condition and yet these individuals appear quite well. On further questioning they will describe the sensation as air hunger and say "I just can't get enough air." During the examination the physician is apt to catch them in the act, so to speak, and see them occasionally take a very deep breath like a sigh. At that very instant it is well immediately to ask the patient if that is what is troubling her. This enables one to identify this primary complaint as functional for the patient actually overventilates the lungs for some peculiar reason and still does not feel that sufficient air is obtained. Even minor changes in the electrocardiogram such as very slight inversion of the "T" waves in any of the four leads and slight depression in the S-T segment have been observed in some cases. When this condition is marked and maintained it may result in sufficient overventilation to produce symptoms of tetany. The patients may complain of tingling and numbness of the extremities, show carpopedal spasm and a positive Trousseau (spasm of the fingers on squeezing the forearm) or Chvostek sign (contraction of the face on tapping the facial nerve). A proper explanation and assurance that nothing serious is going on often is sufficient to cure the patient.

There are other features that are present in these functional cases that deserve attention. Sweating, tremulousness and nervousness together with palpitation make one think of the possibility of hyperthyroidism. At times the differential diagnosis is not simple and will require basal metabolism studies. Although a somewhat increased heart rate is commonly present, if observations are made while the patient is asleep it will be found to be slow. At times the entire picture resembles incipient tuberculosis and constant care should be taken to avoid overlooking a tubercular process by having roentgenograms taken of the lungs when any doubt exists.

Very recently Scherf has reported a group of cases that would ordinarily be classified as cardiac neurosis or neuro-circulatory asthenia, in which he regards the cause to be some disturbance in the endocrine



system. In these cases there may even be minor changes in the electrocardiograms with flat or slightly depressed S-T intervals. Scherf believes these patients can be helped or cured by the administration of estrogenic hormone.

**Neurocirculatory Asthenia.**—The functional cardiac disease that is often designated "neurocirculatory asthenia" is sufficiently important to merit more detailed discussion. This condition, first described during our Civil War, was very prevalent in the First World War. It was interesting to observe the different circumstances under which the symptoms developed. Some of the soldiers first began to show evidence of an unstable neurocirculatory apparatus when they first appeared before the examination boards, when they were not yet drafted into the army. The very thought of becoming a soldier and enduring all the hardships that it entails was sufficient to produce palpitation, weakness, chest pain, tremulousness, sweating, giddiness, and dyspnea. Others first showed these symptoms while they were training in the camps. Still others did well until they were sent overseas. At the other extreme, there were men who carried on sturdily for two or three years, going through terrible experiences in the trenches without showing any evidence of neurocirculatory asthenia and then finally the nervous reserve would become exhausted. As a last straw it might be a simple event which had never previously disturbed them, like a shell explosion, that precipitated the symptoms. It is not surprising that all these different gradations were found during the First World War. If this condition was more frequent among the British soldiers than among the American, and from my own observations I believe this was true, it probably was due to the fact that the British had to undergo the terrific strain of warfare longer than the Americans and that in another year or two we would have had to equip special hospitals to look after these functional cardiacs as did the British.

Probably the most important factor in predisposing to this disease is the constitutional factor. Many have a past history or family history of a subnormal nervous makeup. It is common to elicit a story of nervous breakdown, fainting spells, psychosis, neurasthenia, epilepsy and the like in the past or family history of the patient. With this background it may then be necessary to have a precipitating cause to bring to the surface the symptoms of functional heart disease. The relative importance of those two factors—predisposing and precipitating—will vary in different individuals. No doubt, if one is sufficiently prominent the other need be very slight to produce the symptoms. During the war, when the psychic trauma of combat was most intense and prolonged, individuals without any detectable evidence of a constitutional defect in their nervous stability finally developed "soldier's heart." Contrariwise others with a marked background of nervous instability would break down at the slightest provocation. During the First World War the precipitating causes were linked up primarily with fear and less frequently with infec-



tion. Gunshot wounds, shell explosions, being buried, gassing, "trench fever," rheumatic fever, etc., made up the common direct causes that precipitated the symptoms of neurocirculatory asthenia. In civil life the same hereditary constitutional factors are present but the direct precipitating causes are anxiety following the loss of one's fortune, the death of some friend or relative, a love affair, or any state conducive to fear or emotional and nervous tension.

The symptoms previously enumerated that accompany neurocirculatory asthenia mainly concern the cardiovascular and nervous systems. Because for the most part these symptoms are the very ones that are brought on by effort in normal people, the condition has also been called "effort syndrome." The difference is that in these patients the effort required to produce the symptoms is inordinately slight as compared to that necessary to produce them in normal individuals. Easy fatigability, breathlessness, palpitation and precordial pain are the most common complaints. Those afflicted cannot stand much physical or mental activity. On examination very little of importance is discovered. Some have a peculiar expression of fatigue in their faces. They may show cyanosis of the face and hands which is due to local sluggishness of the circulation. The heart shows nothing abnormal except occasionally a slight systolic murmur. Not only is the heart not enlarged but there is a tendency for it to be small as determined by *x*-ray examination. In most, the electrocardiograms are normal but occasionally slight flattening or even inversion of the T waves in Lead I or II may be found. There may be a tremor of the hands and a moist skin. All that has been said concerning neurocirculatory asthenia or soldier's heart, which is so prevalent in soldiers, is applicable to civil practice, only the cases are less striking.

The importance of this condition is that it must be recognized and not confused with organic heart disease. So often the physician errs and makes a "cardiac cripple" out of one who is structurally sound. A patient with only minor functional complaints may become much worse and even invalided unnecessarily as a result of a mistaken diagnosis. The physician, believing that he is dealing with some valvular or myocardial disease, cautions the patient against overactivity or may even direct that he stay in bed. This convinces the sufferer that his heart is diseased and from then on he becomes more and more introspective and the symptoms become aggravated. It may subsequently become very difficult or impossible for some other physician to convince him that his heart is sound. Having made the diagnosis of functional heart disease one should speak with emphasis and assurance. It is not sufficient to tell the patient that "your heart is normal, but don't overdo." Or in reply to the question "may I play tennis," if the physician says "no, I think that is too strenuous" any intelligent patient has a right to feel that the physician is holding something back from him or is not certain of his ground. One should reply "you can do anything you please; even if you overdo and feel tired no harm will come." From this he may become convinced that there is



nothing to fear and thereby make the most important step in his recovery. Apart from this assurance it is well to try to disentangle any disturbing psychic factors that may be playing a role. All other general hygienic influences conducive to good health should be gone into, such as graded exercise and particularly the question of nutrition. In those who are underweight a gain of weight proves very helpful.

A few words may be appropriate concerning the relation between neurocirculatory asthenia and the present World War. My own personal experience in the last war leads me to the opinion that soldiers with this condition added very little to our efforts as actual fighting men. Once symptoms were present it is unlikely that any were able to carry on front line duty. Some men with milder symptoms who improved under a course of graded exercises were sent back to the trenches. Not many were able to carry on. More often, after a few days of shelling, back they would appear in one hospital after another and occupy bed space for months. The obvious inference is that when the diagnosis is made such a man should not be accepted for military service at all or only for limited duty behind the active front. This policy would be wise until the need for men is so great that second rate soldiers have to be drawn. It must also be appreciated that in making the diagnosis, symptoms are more important than signs, for it is the complaints they have, such as breathlessness, syncope, weakness, palpitation and precordial pain that incapacitate them.

A wrong opinion prevailed amongst most physicians concerning prognosis. It was thought the moment war ended and the fear of suffering or death was over, that the symptoms would all disappear. Follow-up studies showed that not to be the case. Although many men improved, many others retained their symptoms to a greater or lesser extent indefinitely. This could not be explained entirely on the desire these veterans had to receive pensions, for some who did not need or accept pensions continued to be handicapped.

### PROGNOSIS

Prognosis in cases of functional heart disease is uncertain with regard to the symptoms but excellent as to life. Although one does not fear any serious complications and there is no mortality due to this condition, cures are not readily obtained. Often the underlying fear or psychic disturbance cannot be removed or the symptoms go through cyclic changes, improving and then reappearing as different periods of nervous stress and strain come and go. Even without any obvious cause some patients will continue to be bothered by one or another of these functional symptoms as evidence of some neurocirculatory instability. Because of the great fear of heart disease that prevails among the laity, patients with functional heart disease need constant reassurance and encouragement to carry on.



## PAROXYSMAL RAPID HEART ACTION

THE heart action may suddenly become rapid in a variety of ways. There are several different types of disturbances in the heart mechanism responsible for these paroxysms. It is important to distinguish one from the other, because as we shall see later, the treatment and the prognosis differ considerably in the various types. Furthermore, there are occasional instances in which proper treatment must be instituted quickly, otherwise disastrous results ensue. This whole subject, therefore, has many very practical aspects, and it is surprising how intelligent management of these disturbances can be carried out by the use of very simple means.

Paroxysmal rapid heart action comprises those conditions in which the heart suddenly becomes rapid. The termination of such a paroxysm is likewise sudden. If one had the opportunity of observing the actual transition either at the onset or the offset of these attacks, one would notice that the change takes place instantly. A heart rate which is found to be 70 at one moment might suddenly in one second jump to 200 a minute and at the termination of such a paroxysm the rate would fall in one beat from the high level to a normal one. The abruptness of the change is an important point that distinguishes it from a rapid heart in which the mechanism is normal, *i.e.*, normal tachycardia. If a patient is seen with a heart rate of 170 for instance, in which the latter condition obtains, as might be found in surgical shock, hemorrhage, certain fevers, hyperthyroidism and other conditions, it generally will be possible to ascertain that the heart rate rose gradually to this high level. It may have taken minutes, hours or days to change from the normal to the rapid rate. In true paroxysmal heart action no such gradual transitions occur.

The cause of these paroxysms is the inception of a new cardiac mechanism. In the ordinary normal rapid heart the rhythm is really not disturbed. The impulses arise in the normal pacemaker (sino-auricular node) and travel through the heart normally. The only change is that the rate of impulse formation is rapid. In these abnormal paroxysms, on the other hand, the ordinary pacemaker of the heart no longer controls the rhythm and in its place some abnormal mechanism in the auricles or ventricles controls the heart beat. The types of disturbance that may result are paroxysms of auricular tachycardia, auricular flutter, auricular fibrillation and ventricular tachycardia. Although paroxysms may also arise in the a-v node, or the bundle of His and are then called nodal tachycardia, they are rare, of no particular importance, and are impossible to recognize without the use of special apparatus. The following



conditions are also considered from an electrocardiographic point of view in Chapter 21.

### PAROXYSMAL AURICULAR TACHYCARDIA

Paroxysmal auricular tachycardia is the most common of these disorders. It occurs much more often in patients who have no heart disease than in those with organic disease. In this sense it may be regarded as a functional disturbance. The heart rate during an attack will generally range between 150 and 250 a minute. The attack begins and ends abruptly. It may last minutes, hours, days or rarely weeks. During the paroxysms the heart rate is perfectly regular and the sounds are all alike in quality and intensity. The regularity is so precise that on careful measurement contiguous heart cycles will not differ in length by more than  $\frac{1}{100}$  second. The rhythm is not only regular but seems to be constant and fixed at the same level for long periods at a time. If an attack were to continue for some hours the heart rate might be found to be 196 at one time and ten minutes later it would still be 196. It is also impossible to disturb this rate by such simple means as having the patient hold his breath or change his position, which measures generally will alter the rate of a normally beating heart. This constancy of rate is an important distinguishing characteristic which enables one to recognize the condition at the bedside. To obtain this the apex rate should be counted accurately for sixty seconds. This can be done with a stethoscope in human beings, when the heart is regular, even when the rate is as rapid as 250 a minute. The error in the count need not be greater than two, one at the beginning and one at the end of the minute while trying to synchronize the heart beat with the second hand of the watch. During the intervening beats there need be no inaccuracy whatever. Accurate counting of a rapid regular heart may be facilitated by simultaneously tapping with the foot or the finger. This matter deserves some emphasis because one often hears the expression that the heart was too rapid to count. If the rhythm was regular this generally means that the observer made no real attempt to count accurately. Let us assume that in a given case the rate of 164 was found. Some minutes later the count should be repeated having the patient change his position, hold his breath or go through any maneuver which ordinarily disturbs the heart rate. On the recount the rate will again be found to be 164 or 163. If the second rate were 170 or 160 one might fairly safely assume that the patient did not have paroxysmal auricular tachycardia. A normally accelerated heart, however, might alter its rate to that extent. Thus we have a simple means which helps to characterize this condition. Expressed in other terms, while ausculting the heart not even the slightest alterations in the rhythm will be noted as a result of those measures which generally produce acceleration or retardation under normal circumstances.

**Symptoms.**—The clinical features in this condition are variable. The attacks may occur rarely, one every several years, or frequently. I have



seen instances in which the patient had as many as ten paroxysms every day. He will generally complain of sudden palpitation of the heart and become nervous and agitated. Fainting and actual loss of consciousness may occur at the onset of any form of paroxysmal rapid heart action. This probably results from the sudden decrease in the cardiac output with resulting relative cerebral anoxemia. Adjustments in the circulation take place, however, so that syncope does not last very long. During the first time or two, not knowing what it all means, he may actually fear that he is going to die. Usually there is no dyspnea with the paroxysm and after a variable length of time the attack ends as suddenly as it commenced. There frequently is a sensation of gaseous distention of the abdomen or belching of gas during and particularly at the end of the paroxysm. Occasionally a cessation of the attack comes with a vomiting spell. These features make the patient think that the paroxysm is due to "indigestion" or that it is in some way related to his diet. The causes that precipitate the attack are numerous and inconstant. Frequently a sudden movement of the body such as bending to tie one's shoe or a quick turn of the head initiates the attack. Much less frequently violent effort brings on a spell. Emotional factors also come into play. Sudden thoughts or even dreams may be the precipitating cause. There remains a great number of instances in which the attack seems to occur spontaneously without any known cause. In some cases there is heart pain during a paroxysm which is more apt to occur after the paroxysm has lasted some time. In its description it may resemble the pain of angina pectoris but it has by no means the same prognostic significance. For, when the heart rhythm is normal and slow there will be no chest pain whatever and in fact no other evidence of heart disease.

Physical examination of the patient during an attack is apt to reveal no essential abnormality except for the rapid heart rate. The patient may seem pale and agitated and the skin may be moist. When an attack develops in one who is otherwise normal there will be no evidence of circulatory insufficiency except in very rare instances. This is also true in some cases in which there is associated organic heart disease, either valvular or muscular. In fact there is no other condition in which the heart may be so rapid with so little apparent embarrassment to the circulation. The patient may have no dyspnea or cyanosis and yet show a heart rate of 200 or more.

Under certain circumstances symptoms of considerable gravity develop, such as peripheral thromboses and congestive heart failure. The factors that determine the development of these complications are the following: the duration of the attack, the heart rate during the attack and the condition of the heart before the onset of the attack. It is obvious that a normal heart might stand the rate of 240 a great deal longer than a heart with mitral stenosis, or one with a poor degree of compensation. I have seen a patient with mitral stenosis develop marked dyspnea, edema of the lungs, cyanosis and engorgement of the liver within a few



hours after the onset of tachycardia in which the heart rate was 190. On the other hand, if the rate is extremely rapid and the attack of long duration, even if the heart is structurally normal, disastrous results may develop. This was well illustrated in a case I observed many years ago. A man of forty who was otherwise perfectly well had had three attacks of tachycardia during the previous few years. Each attack lasted uninterruptedly for five to eleven days. During the first one he developed a hemiplegia from which he gradually recovered in the course of a few months with a slight residual spasticity of one side of the body. During the second attack he developed aphasia which gradually disappeared in four months. During the third attack, dry gangrene of the left arm developed requiring amputation at the shoulder. I saw this man in 1914 during the fourth attack and found the heart rate to be 250 a minute. After obtaining certain data on this patient the attack was immediately ended by ocular pressure. It was the first time his attacks had been controlled, for the others had stopped spontaneously after lasting many days. In fact this may have been the first instance in which ocular pressure was ever effective as a treatment for paroxysmal tachycardia.

The explanation of the complications that occurred in this case was quite obvious. While the heart was beating 250 times a minute the pulse pressure was extremely low. During several attacks in which the patient was observed the systolic pressure was around 94 to 96 and the diastolic around 88 mm. Therefore, this patient had an effective pulse pressure of no more than 6 or 8 mm. One can readily see from this how thrombosis in peripheral vessels could easily develop. This must have occurred in the cerebral vessels during two of the attacks and in the vessels of the left arm at the time gangrene occurred. The process essentially consisted of stagnation of the blood. In such a case it is evident that proper therapy was imperative, for this patient is still alive and in fairly good health.

In general, the changes in blood pressure that occur during paroxysmal tachycardia consist in a tendency for the systolic level to fall, and the diastolic to rise with a diminution in the pulse pressure. The degree of such changes will depend upon the three factors mentioned, namely, the heart rate, the duration of the attack and the structural condition of the heart. Such attacks have at times been called acute dilatation of the heart. This is a misnomer, for in a series of twelve such instances *x*-ray measurements of the heart before, during and after attacks failed to show an appreciable dilatation except in one instance (case cited in the preceding paragraphs in which the heart rate was 250). In some patients the heart was actually smaller during the attack. Other interesting findings that may develop during attacks of tachycardia are a slight fever and a slight leukocytosis. I do not think that these features indicate infection. They are rather the result of congestion.

One can readily see how a simple attack of any type of paroxysmal rapid heart action may be confused with acute coronary thrombosis. A patient has a "heart attack." The heart rate is found to be very rapid.



There may be some collapse, a fall in blood pressure and even chest distress of the anginal type. Fever and leukocytosis may follow. After the attack has ended the electrocardiograms may even show inversion of the T waves suggesting coronary thrombosis. These changes are probably the result of myocardial fatigue and local relative anoxia and gradually return to normal in subsequent days or weeks. All this can occur in a structurally normal heart and also leave the heart entirely intact. The confusion becomes even greater when one considers the possibility that actual coronary thrombosis may result in vulnerable individuals if a low blood pressure and a state of shock persists for a long time. One important distinction is that in simple paroxysms of tachycardia the very rapid rate comes first and then the above signs and symptoms follow, while in coronary thrombosis the abnormal tachycardia follows the attack and generally does not appear for one to several days.

**Treatment.**—There are certain circumstances in which the development of paroxysmal tachycardia may present a serious situation and require immediate and effective treatment. I have reference to its occurrence during labor or during a surgical operation. I recall an experience in which a woman was being operated on for gallstones. While she was under ether, just as the operation was to be started, respiration ceased, the radial pulse became imperceptible and extreme cyanosis quickly developed. Artificial respiration had to be instituted and had already been carried on for about ten minutes when I saw the patient. The heart rate was 212 and perfectly regular. Carotid sinus pressure stopped the attack immediately, the heart returning to a normal rate of 80. Normal breathing was immediately restored and the cyanosis disappeared. The patient was subsequently operated on and the gallbladder containing stones removed. In two other cases that were somewhat similar but in which the symptoms were not so alarming, carotid sinus pressure was not effective whereas ocular pressure promptly restored the heart to its normal rate. Such experiences are not common. The urgency and gravity of the whole situation is so great when they do occur that they deserve this emphasis.

There are two phases to the treatment of paroxysmal tachycardia; the first concerns the treatment of the attack and the second the procedures that may prevent or diminish the incidence of future attacks. There are a great many measures which have seemed to be successful at one time or another in stopping attacks. They all produce their beneficial effects by stimulating the vagus nerve in one way or another. The act of vomiting is one of these, either produced spontaneously or by the use of an emetic, such as apomorphine, ipecac, and the like. One to 4 teaspoonfuls of syrup of ipecac often is effective when other methods fail and may be repeated in one-half to one hour. I knew one patient who stopped several of his attacks by drinking ice-cold water or swallowing bits of ice. Occasionally lowering the head or lifting the foot of the bed has proved successful. One simple and most helpful measure is holding a deep breath. There



are many patients who have found that this simple experiment can stop their attacks very effectively. It is well to tell such patients to take a deep breath and hold it as long as they can. In normal individuals, holding a forced inspiration produces a slight vagal slowing of the heart, but in this condition it will frequently bring an attack abruptly to an end with restoration of a normal slow rhythm. (Valsalva experiment.)

Finally, there are three methods which stimulate the vagus nerve more effectively. The first of these is by irritation of the carotid sinus, the second by pressure over the eyeball and the third by the use of drugs. The first of these procedures consists of palpating for the bulge in the common carotid artery as it divides into external and internal branches with one hand while supporting the patient with the other hand placed behind the neck. Pressure is then exerted backward, completely compressing the artery and massaging it for several seconds. This should be done first on one side of the neck, then on the other, but not on both sides at the same time. The second method consists in exerting rather firm pressure with the thumb over the eyeball. This will be painful. Here again one eye at a time should be tried. This pressure sends a stimulus up the fifth cranial nerve and by a reflex (oculocardiac reflex) down the vagus nerve. When ocular pressure is effective it is not as a result of the pain, for pain produced elsewhere in the body causes no such slowing, and I have several times stopped attacks while a patient was under anesthesia. Its use is limited to some extent but there are instances when this will prove successful after other methods have failed.

Attacks of auricular tachycardia can generally be arrested by one or another of the procedures just described. There are instances, however, when the tachycardia is resistant to all these measures. Drugs may then be used which have proved valuable in stubborn cases. On several occasions in which the tachycardia persisted for a long time and could not be stopped by the ordinary means and the patient's condition seemed rather critical, the intravenous injection of quinidine sulfate immediately restored the normal rhythm. For this purpose one would generally need 0.3 gram of quinidine sulfate. It is best to inject this slowly while someone is listening to the heart over the precordium. Just as soon as the break in the rapidity occurs, injection should be stopped. In two cases I found that after 0.2 gram of the drug was injected, and while the hypodermic needle was still in the vein, the attack suddenly came to an end. There is some danger in the use of quinidine, and, therefore, this treatment should not be used until other measures fail and the patient's condition is critical enough to warrant it. Another drug that has been used with great success is acetyl- $\beta$ -methyl-choline. The average adult dose is 20 milligrams given subcutaneously. Infants have responded favorably to doses of 5 milligrams. This is a powerful vagal stimulant. Occasionally untoward results occur, particularly in allergic individuals, for which  $\frac{1}{80}$  to  $\frac{1}{30}$  grain of atropine should be used. Other drugs have been used successfully to stop attacks. Amongst these are calcium gluconate (10 to



20 c.c. of 10 per cent solution given slowly intravenously), parathormone subcutaneously, magnesium sulfate (15 c.c. of 20 percent solution intravenously) and prostigmine methyl-sulfate (0.5 to 1.0 milligrams intramuscularly). There is reason to believe, however, that calcium might be dangerous in ventricular tachycardia. Curiously enough, I observed one patient who had typical angina pectoris and in addition classical attacks of paroxysmal auricular tachycardia. The two conditions were independent of each other. This patient found that nitroglycerin not only relieved the attack of anginal pain (when the heart rate was normal) but stopped his attacks of tachycardia. Finally large doses of digitalis (0.5 to 1.0 gram) may prove successful occasionally.

It must be understood that in most cases of paroxysmal tachycardia the attacks eventually cease spontaneously and that the patient is none the worse for the spell. It is only occasionally that any heroic treatment would be indicated, but there are instances in which it might prove life-saving.

When it comes to preventing the return of such attacks, until recent years there was practically no treatment that was effective. The patients themselves often feel that if treatment were directed at the gastrointestinal tract they might be cured. The physician frequently alters the diet in one way or another, advises medical treatment for gas, indigestion or constipation, with the hope of preventing these attacks. In other instances courses of sedatives have been prescribed to diminish the general nervous irritability. In those cases in which there is excessive use of tobacco this may be ascribed as the cause of the attacks. Treatment along these lines, however, has always proved practically useless. The two drugs that are helpful in preventing recurrent tachycardia are quinidine and digitalis. Either one or the other, if properly administered, will often either inhibit the attacks entirely or will diminish their frequency and severity. In several instances I have found digitalis to be effective after quinidine had failed. In only a portion of the cases is this treatment indicated. If attacks come only on rare occasions, such as once in six to twelve months, it would hardly seem wise to prescribe a course of preventive treatment. Not knowing when an attack is to be expected, such a patient would have to be constantly under the influence of quinidine or digitalis in order that the heart might be prepared on that distant day when the attack otherwise would be due. This would necessitate the constant use of a medicine for the possible prevention of a rare attack. Even if months elapsed and the patient were free from attacks, because of the infrequency of such spells, it would still remain doubtful whether the therapy had anything to do with the result obtained. Under these circumstances it is best to explain the entire situation to the patient assuring him that nothing serious will develop, to institute no constant drug therapy, but to be prepared to meet each spell as it arises.

When the attacks occur frequently, treatment should be specifically directed at their prevention, for then they may be actually incapacitating



and it becomes a simple matter to decide whether or not therapy is effective. Quinidine sulfate may be given in doses of 3 to 5 grains three times a day. If the patient ordinarily has attacks every few days a particular dose should be continued long enough to be certain whether it is effective or not. If attacks continue despite this dose it should be increased. Ten grains three times a day can be tolerated over long periods of time. Although some patients may tolerate larger doses without showing toxic symptoms, such as ringing in the ears, as a practical matter if attacks continue while 5 grains three times a day are taken it is best to omit the drug entirely.

It was formerly thought that digitalis was of no use in the treatment of paroxysmal tachycardia. This opinion was held mainly for two reasons. In the first place it seemed true that the ordinary administration of digitalis during an attack had little influence in stopping it. In the second place its use in preventing the recurrences seemed disappointing because complete and constant digitalization was generally not carried out. Some years ago I had an opportunity of observing four patients who had had courses of quinidine and digitalis therapy without success. The digitalis in these cases was inadequate and when the dosage was administered in quantities similar to those used in heart failure, all attacks ceased. One of these patients was having several attacks a day so that he had to quit his work. He had no organic heart disease and except for these attacks he was well. Under full doses of digitalis all attacks ceased and he was able to return to work. Another patient was a woman aged sixty who had had attacks about every two weeks for the previous three years. They grew increasingly severe so that finally they were absolutely prostrating. When she was seen during one of these spells she was found to be pulseless, no blood pressure reading could be obtained and the heart rate was 240. She was cold and it looked as though she might die. Ocular pressure instantly brought the heart rate to normal. She had previously taken quinidine at one time, and at another 5 drops of digitalis three times a day for some months without success. She had also taken sedatives and various medicines for indigestion. After the spell just described she was given  $1\frac{1}{2}$  grains digitalis leaves three times a day for one week and thereafter  $1\frac{1}{2}$  grains daily. There were no attacks for fifteen months. It was then decided to stop digitalis to see whether or not it was needed any longer. About one week after the drug was omitted a similar attack occurred and digitalis was then reinstituted and the patient had on further attacks. This case was a very striking one for the patient remained perfectly well for over fifteen years, whereas formerly for three years she was in constant fear of these attacks, and had to live a very confined life and required constant nursing attention.

In the more common type of paroxysmal auricular tachycardia just described, there is no heart block, the ventricles responding to every auricular impulse. There is a much rarer variety in which block does occur. In this and other ways this type resembles auricular flutter to



some extent. It will not respond very readily to the methods of treatment just mentioned. Quinidine or digitalis may be tried, however, for occasionally success may be obtained. It is desirable at least to keep the ventricular rate slow by constant use of digitalis, if possible, for the rapid auricular rate may persist for weeks or months. New observations by Wilson and his associates have presented evidence suggesting that this type of auricular tachycardia and possibly others as well are due to a circus movement in the auricles, in which the pathway goes through the a-v node.

### PAROXYSMAL AURICULAR FLUTTER

Another form of paroxysmal heart action is auricular flutter. Although this condition once established is apt to be persistent, occasionally it occurs in paroxysmal form. Here the auricular rate generally ranges from 250 to 350 and the rate of the ventricle is one-half of the auricular rate or less. In very rare instances the ventricular and auricular rates are the same. When the condition is first seen, before any treatment is instituted, there is apt to be a 2:1 heart block, so that whatever the auricular rate may be, the heart rate as counted at the apex is half as great. The former may be 340 per minute whereas the ventricular rate is 170. It should be borne in mind that the rate of the auricle is really inferred, for the auricular contraction cannot be heard. The rhythm in untreated patients is generally regular, because every second impulse coming from a regularly beating auricle reaches the ventricle. Occasionally in untreated cases and frequently after treatment has been started the rhythm is irregular. Here the degree of block is changing in different heart cycles, so that for a short time every second auricular beat gets through to the ventricles, then only the third or fourth beat gets through and so the regularity is disturbed from time to time. The resultant irregularity may occasionally be gross enough to resemble auricular fibrillation very closely. There may even be an appreciable pulse deficit. The essential difference between the two conditions is that in auricular fibrillation the irregularity is actually complete whereas in flutter it follows some definite law, *i.e.*, different groups of ventricular beats will have the same length if accurately measured and properly selected, as they will correspond to the same number of regularly occurring auricular cycles (Chapter 21, Fig. 32).

This type of paroxysmal rapid heart action occurs generally in patients who have organic heart disease, either valvular or myocardial, but may also occur in those with no other evidence of heart disease. Unlike paroxysmal auricular tachycardia it is apt to persist for long periods at a time or even to remain permanent if untreated. Therefore, it is important to try to restore the normal mechanism or at least to control the ventricular rate. There are two purposes in the treatment of patients with this condition. It would be desirable to do away with the mechanism of auricular flutter entirely or to prevent the ventricles from becoming



rapid if the flutter persists. In general the two drugs that are used for these purposes are digitalis and quinidine. In about one-third of the cases if digitalis is properly given the heart is restored to normal rhythm. At first, as has been mentioned, the ventricular rate is apt to be rapid and regular with a coexisting 2:1 heart block. After the customary dose of digitalis is administered the ventricular rate slows and may become irregular as a result of an increase in heart block so that a 4:1 or 6:1 a-v block results. If the ratio between ventricular and auricular beats is constantly 4:1 or 6:1, the rhythm is regular, if this ratio is changing in different cycles the rhythm is irregular. The ventricular rate may now be 60 or 80 but the auricular rate during this time remains unchanged, as the flutter so far is not disturbed. At this point in some cases, auricular fibrillation may suddenly develop with the appearance of a gross irregularity. If digitalis is now omitted, during the course of the next day or so the heart may return to a normal rhythm. In general, therefore, digitalis may change the auricular flutter into auricular fibrillation and permit the auricles spontaneously to return to a normal rhythm. When this treatment is ineffective, auricular flutter may persist even though the ventricular rate has been slowed by digitalis, or having produced a change to auricular fibrillation this may continue indefinitely, or again return to the original state of flutter. When digitalis does not restore the normal rhythm it may still serve an important therapeutic purpose in maintaining a slow ventricular rate, whether the original flutter or the subsequent fibrillation persists. Inasmuch as the most important purpose of treatment is the prevention of a rapid ventricular rate, no matter what the auricles are doing, digitalis is of distinct value in this condition.

The best treatment of auricular flutter is actually to restore a normal rhythm. It is known that quinidine increases the refractive period of auricular muscle. By so doing it may actually stop the continuous circus which exists in the auricle and which tends to perpetuate the flutter. The dose of quinidine for this purpose is not fixed. In general, from 5 to 10 grains three to four times a day may be given. The procedure I have used is, after digitalis has slowed the ventricular rate and the abnormal rhythm persists, to start with a dose of 0.2 gram and to increase it by 0.1 gram with each dose, giving the medication three times a day. During the quinidine therapy a daily dose of 0.1 gram of digitalis may also be given. At times I have had to increase the single dose of quinidine to 1.0 or even to 1.5 grams before reversion to normal rhythm was obtained. It is desirable to have the patient under close observation during this time so that frequent electrocardiograms may be taken. It may be noted that during the quinidine administration the actual auricular rate will slow while the ventricular rate accelerates. I have seen the auricular rate of flutter drop from about 300 to 150 as a result of quinidine. At any time during this treatment the normal mechanism may suddenly be restored. The drug is by no means effective in all cases, and unlike digitalis has no value in slowing the ventricular rate except when it restores the



normal rhythm. The disadvantages of quinidine apart from its frequent ineffectiveness are that it has certain toxic actions that occasionally are serious. On the other hand there are instances of auricular flutter that are helped greatly by this drug when digitalis has been ineffective.

### PAROXYSMAL AURICULAR FIBRILLATION

Paroxysmal auricular fibrillation is a much more common condition than it was thought to be not so long ago. In fact the term "auricular fibrillation" is applied to a condition that was formerly called "perpetual arrhythmia." It was then believed that once this became established it remained so permanently. We now know that the transient form is quite common. It occurs occasionally in patients with mitral stenosis before it takes on the permanent form. It is also frequently met with in hypertensive heart disease or in so-called "chronic myocarditis." It occurs in some cases of rheumatic fever and in a small proportion of cases of pneumonia. It has been frequently observed during acute coronary thrombosis. Rarely it is seen under a variety of circumstances that seem to have no very definite relations to the heart, *e.g.*, acute angioneurotic edema, chronic gallbladder disease, as a complication of any surgical operation requiring general anesthesia, especially in operations on the lungs and mediastinum, etc. Possibly the most common condition with which transient auricular fibrillation is associated, is hyperthyroidism. This is a frequent event during the ordinary course of the disease and particularly during the first day or two following an operation on the thyroid gland. There remains a small group of patients in whom transient fibrillation occurs and in whom there can be detected no other evidence of heart disease or disease of any important organ. It must be regarded in these isolated cases as a purely functional derangement and not indicative of any disease. It is evident from the great variety of conditions in which transient fibrillation may occur that the symptoms resulting from this disorder and the appropriate treatment must vary a great deal. It is obvious that the sudden inception of a rapid irregular rate can produce very distressing symptoms in a patient who already has serious organic heart disease, whereas if it occurs in a heart that is essentially sound very little embarrassment may result. As will be seen later, in some cases this paroxysmal arrhythmia needs no treatment whatever or treatment may be entirely useless until the underlying cause is removed, whereas in other instances the attack itself presents a therapeutic problem of major importance.

When auricular fibrillation develops the rhythm of the heart becomes grossly irregular. Almost invariably the rate as counted at the apex is quite rapid, *i.e.*, 130 to 170 or more. If the patient is digitalized or if, as rarely happens, there is a concomitant defect in the conduction apparatus the apex rate may be slow. With a rapid heart rate there generally is an appreciable pulse deficit, *i.e.*, the pulse rate is 10 or more less than the heart rate. Occasionally when there is hypertension or when the pulse



pressure is great, as in hyperthyroidism, or when the heart rate is not very rapid there may be very little if any pulse deficit. The bedside diagnosis, however, in the great majority of cases is not very difficult. The most important feature in the diagnosis is the character of the irregularity. It is a complete, absolute and tumultuous arrhythmia that one hears. Only in rare instances may it be confused with other conditions.

The symptoms that result from an attack of auricular fibrillation depend in a great measure on the state of the circulation before the onset of the attack. In most cases there is palpitation. This is produced by the rapid agitated contractions of the heart. If there already exists cardiovascular disease, dyspnea of a mild or marked degree may quickly develop. In fact some cases may present the picture of acute pulmonary edema. Occasionally an attack of transient auricular fibrillation is associated with the dislodgment of an embolus from an auricular thrombus. If the thrombus is dislodged from the right auricle a pulmonary infarct develops and if from the left auricle hemiplegia or other arterial infarction may occur. If auricular fibrillation develops in a patient already very sick with pneumonia sudden collapse may occur. I recall one such case in which the heart rate suddenly rose to about 200 and the pulse became imperceptible. The patient seemed to be *in extremis*. Twenty minutes after an intravenous dose of strophanthin he quickly revived, the heart rate fell to about 110, the pulse returned and the patient eventually recovered. There are other instances of transient fibrillation in which the circulation is in such good condition that the patient will have no complaints whatever, except a slight degree of palpitation. One can readily see how varied the picture may be.

**Treatment.**—There are two therapeutic aspects to this condition; one concerns itself with the underlying disorder whether it be pneumonia or exophthalmic goiter, etc., and the other is the treatment of the specific attack of fibrillation. In hyperthyroidism it is useless to treat the patient for auricular fibrillation in the ordinary way, as attacks will tend to recur as long as the hyperthyroid state persists. When the treatment of hyperthyroidism is effective in restoring the metabolism to a normal level the tendency to fibrillation will generally spontaneously disappear. In most other types of transient fibrillation direct therapy for the arrhythmia itself is indicated. The two drugs that have distinct therapeutic value in the treatment of this arrhythmia are digitalis and quinidine. The action and the purpose of these two drugs are quite different and so are the results obtained from their use. Digitalis rarely brings an attack of auricular fibrillation to an end. When such a spell ceases while the patient is taking digitalis it is thought to do so spontaneously. In fact there is reason to believe that digitalis might tend to perpetuate the condition. The value of digitalis, however, is to slow the ventricular rate while the fibrillation continues. Contrariwise, quinidine may even cause an acceleration of the ventricular rate while fibrillation is present, but its main value lies in the fact that it tends to do away with the arrhythmia entirely and



restore the heart to a normal mechanism. If it is known that the patient has had previous spells of this arrhythmia of short duration lasting only a few hours, and the general state of the circulation is quite satisfactory, it is just as well to give neither one nor the other form of medication, but rather to administer some sedative or possibly morphine, for in a few hours the attack may be over. If, on the other hand, the patient already has sufficient organic cardiac disease so that the rapid irregular ventricular rate has already produced or may readily produce generalized congestion or alarming symptoms of circulatory insufficiency, rapid digitalization is indicated. The reason for this is that it cannot be known how long the attack is going to last or whether or not the fibrillation will become permanent. Whether digitalis shall be given by mouth, intramuscularly or intravenously depends upon the urgency of the situation. The following is an illustration of this problem. A middle-aged woman with fairly well-compensated mitral stenosis had, during the course of several years, a few transient attacks of auricular fibrillation, each lasting about an hour or two. During these years dyspnea had been slowly increasing. Finally she had a spell which had already lasted about five hours when I saw her. In this brief time her condition had become alarming. She was semistuporous, had marked acute pulmonary edema, striking generalized cyanosis and extreme dyspnea. The heart rate was about 190 and the pulse was practically imperceptible. The situation seemed desperate. Inasmuch as she had not been taking digitalis, 8 c.c. of digifolin (0.8 gram digitalis) were immediately given intravenously. In about twenty minutes, although the auricular fibrillation still continued, the heart rate fell from 190 to about 100. The change in the appearance of the patient was most dramatic. The dyspnea, cyanosis and pulmonary edema all quickly improved and she fell back into a comfortable sleep. As it happened, fibrillation from then on persisted. She continued on digitalis by mouth and later became ambulatory. To be sure, in most cases no such alarming symptoms result. In general, palpitation is the prominent complaint and under such circumstances digitalization by mouth is sufficient. In a case of moderate severity 0.5 gram of digitalis may be given by mouth in one dose and if none has been previously given, this same dose may be repeated in several hours and again the following day if the heart rate is still rapid. Such a procedure will be effective in relieving the symptoms of circulatory insufficiency. It is evident from the foregoing discussion that digitalis should be used to slow the ventricular rate when that rate is rapid, when the attack seems to be persisting and when embarrassment to the circulation results.

The second phase of the treatment of paroxysmal auricular fibrillation is the prevention or the diminution in the frequency of further attacks. In most cases it is best to try the effect of quinidine sulfate. This may be given in doses of 0.3 gram three times a day. It should be continued for a sufficient time to ascertain whether attacks are being inhibited or not. If they had been coming once or twice a week and if while the patient



was taking quinidine none had occurred for several weeks, it is fair to say that the attacks were specifically inhibited by quinidine. The drug might then be continued for several months. If, however, the attacks persisted with equal frequency on this dose, it generally means that quinidine was of no value, although a larger dose might be tried. Occasionally attacks have a tendency to recur at certain times of the day or night. In these cases the dose of quinidine may be given an hour or so before the expected attack and prove helpful. If quinidine sulfate is ineffective as a preventive and the attacks are troublesome or frequent, the patient should be digitalized thoroughly and kept on maintenance doses constantly. This need not prevent the return of the spells, but will so prepare the heart that when they do occur the ventricular rate will not be rapid. In one such case when the attack occurred before digitalis was given the heart rate was about 140. A few days later after only 1 gram of digitalis had been taken by mouth a similar attack occurred and the heart rate was about 100. The following week when the patient was completely digitalized, on routine examination the rhythm, which previously was regular, was found to be grossly irregular with a rate of 70. The patient was entirely unaware that she was having a spell, whereas previously spells caused considerable palpitation. This experience clearly illustrates the effect of digitalis, for the spells continued but the resultant symptoms were obviated. It is also a good example of the slowing effect of digitalis on the ventricular rate in the absence of heart failure, for there was no evidence of congestion in this case. There will be occasional instances in which digitalis may be used in the treatment of one specific attack and later quinidine could be given to prevent recurrences. It must be borne in mind that many spells pass off without therapy.

### PAROXYSMAL VENTRICULAR TACHYCARDIA

The last type of paroxysmal rapid heart action to be considered is paroxysmal ventricular tachycardia. In this condition the beats arise in the ventricle itself and the auricles either contract at a different rate or in some instances there is reversed conduction so that the impulse that arises in the ventricle travels upward to make the auricles contract. There are several important differences between this type of tachycardia and those arising in the auricles. This condition in the great majority of cases is associated with serious structural heart disease, generally coronary thrombosis. There are occasional instances of its occurring with chronic valvular heart disease and very rare cases in which the heart is structurally normal. Unlike auricular tachycardia, which is often unassociated with any serious heart disease, it may generally be assumed that ventricular tachycardia indicates a serious heart disorder.

It formerly was thought that one could not possibly distinguish at the bedside the ventricular from the auricular type of tachycardia. It was considered necessary to have electrocardiograms to identify them. During recent years, however, some simple clinical criteria have been dis-



covered which enable one to make fairly accurate diagnoses using only those means that every physician can readily employ in the sick room. It has been emphasized that in auricular tachycardia the rhythm is perfectly regular. There are no interruptions in the rhythm. In fact the differences in length of contiguous heart cycles, if measured accurately, would not be more than one-hundredth of a second. In ventricular tachycardia, although the rhythm may seem to be absolutely regular for stretches of many seconds or even of minutes, one may detect, on careful auscultation, in most cases, occasional irregularities. Secondly, in auricular tachycardia the heart sounds of the consecutive regular rapid beats are all alike, whereas in ventricular tachycardia every now and then the first heart sound will vary in intensity or quality. It will become accented or muffled or reduplicated. This is due to the inconstant relation between the time of ventricular and auricular systole. Another distinguishing feature is the jugular pulse. Here one may see fewer auricular beats than the ventricular rate as counted over the precordium and the jugular waves are often prominent because the auricles may be contracting while the ventricles are in systole. Finally, the various measures employed to stimulate the vagus nerve which may end an attack of auricular tachycardia will never influence the rhythm of ventricular tachycardia. These criteria together with the general clinical differences such as the association of the one with functional heart disease and of the other with grave coronary disease should make it possible to recognize ventricular tachycardia fairly accurately even without special apparatus.

The beginning and ending of an attack of ventricular tachycardia are abrupt and instantaneous just as in other forms of abnormal rapid heart action. The rate during the attack is usually between 150 and 200 but occasionally a higher rate is reached. When it occurs without organic heart disease these individuals may complain merely of palpitation although even this may be incapacitating. When, as more frequently happens, it develops during the days that follow an attack of acute coronary thrombosis, it presents a serious complication of an already grave condition. This complication may of itself prove fatal. Proper recognition of the condition becomes very important inasmuch as treatment may be effective in restoring the heart to a normal rhythm.

**Treatment.**—It has just been mentioned that measures like carotid sinus pressure or ocular pressure are ineffective in arresting ventricular tachycardia. Likewise, digitalis has no beneficial effect on this condition. In fact, there is evidence that digitalization may tend to prolong such attacks and accelerate the rate further. I studied one instance in which the heart rate rose from 145 to 180 as the patient was given full doses of digitalis. The drug that can restore the heart to a normal mechanism when ventricular tachycardia is present is quinidine. There is reason to believe that the mechanism involved in the condition is a circus movement just as it is in auricular fibrillation and auricular flutter, only in the former the circus is in the ventricles and in the latter it is in the auricles.



It is logical to infer that the action of quinidine in breaking up this circus is likewise the same in the two conditions. It has been my experience that quinidine has been effective in a larger proportion of cases of ventricular tachycardia than in those of auricular fibrillation.

The therapeutic dose of quinidine in this condition is variable and has to be determined individually in different cases. One patient who had no organic heart disease but complained of frequent attacks of palpitation, which were proved to be due to ventricular tachycardia, remained entirely free from attacks as long as she took 0.3 gram of quinidine three times a day. Other instances, in which the attacks occurred during coronary thrombosis, were controlled by doses of 0.4 and 0.6 gram three times a day. One patient with mitral stenosis, auricular fibrillation and ventricular tachycardia required 0.8 gram to stop the tachycardia and doses under 0.8 gram three times a day failed to prevent a return of the condition. He maintained a normal rhythm for many months while taking the larger dose (0.8 gram). Finally, one very striking case of coronary thrombosis in which this treatment was actually life-saving required 1.5 grams five times a day for several days, both to stop an attack and to prevent its return, smaller doses having proved to be ineffective. In the last instance we dared to give such large doses because the condition of the patient seemed otherwise hopeless and it was found that smaller doses had had a partial effect in slowing the ventricular rate from 200 to about 150. This slowing of the ventricular rate on increasing doses of quinidine is almost a constant finding. At times the rate keeps slowing with each large dose, only to return to the original level as the effect of the drug wears off. On rare occasions a large dose of atropine given subcutaneously while the rate was partially slowed by quinidine promptly restored the heart to a normal rhythm. In selected cases quinidine may be given intramuscularly or even intravenously. There are now available ampules of quinidine for parenteral use. The physician will have to weigh the circumstances carefully before employing methods of therapy that entail some risk. It is, therefore, evident that the judicious use of quinidine in ventricular tachycardia may be of considerable value and on rare occasions life-saving.

Magnesium sulfate (2.0 to 4.0 grams, intravenously) may also stop ventricular tachycardia instantly. I have seen patients in whom the attack stopped within one minute. In others it failed when quinidine administered intravenously was successful.

#### DIAGNOSIS OF TYPES OF PAROXYSMAL RAPID HEART ACTION

As an aid in the diagnosis of the various types of paroxysmal rapid heart action a composite figure of the effect of vagal stimulation on these arrhythmias has been constructed (Fig. 35). A study of this figure will enable the physician to visualize the results obtained by simple physical examination. The first tracing shows the effect of right carotid pressure in a normal heart. There is a gradual slowing and a smooth return to



the original rate. The second curve shows a similar slowing effect when normal tachycardia is present. This patient had an acute coronary thrombosis and the rapid heart rate made me wonder whether he had flutter; in fact the appearance of the electrocardiograms on first glance suggested this diagnosis. The gradual slowing of the heart with a smooth return to its original rate eliminated this diagnosis. The third case illustrates a typical arrest of an attack of auricular tachycardia. One observes the abrupt cessation of the rapid rate with a prompt resumption of normal beats. Interruptions during the transition due to extra systoles or vagal effects are common. No other type of rapid heart action is completely controlled in this fashion.

The fourth tracing shows the effect of carotid pressure in auricular flutter. The ventricular rate is promptly slowed (although the auricular rate remains unaffected), but there follows an "irregular" return to the previous rapid rate. The point that distinguishes flutter from normal tachycardia is that, as the vagal effect subsides, the ventricles return to their original rate in a jerky fashion. After a short cycle a longer one may appear, then another short one, until finally the constant rapid rate is resumed. This is to be compared with the second tracing where it may be seen that each beat becomes shorter and shorter until the original rate is restored. The fifth set of electrocardiograms illustrates the events in auricular fibrillation. The rhythm is grossly irregular at the start, becomes slower during the vagal stimulation and then returns to its original absolute arrhythmia. The final curves are those of ventricular tachycardia. Here no effect whatever is produced. Very slight irregularities which are fairly characteristic of this condition continue throughout the tracing.

When the changes that have been described are translated into corresponding auscultatory findings obtained over the precordium, it is striking what accurate diagnoses can be made by simple bedside methods.



## 14

### ACUTE CARDIOVASCULAR EMERGENCIES

A PRACTICING physician is often confronted with apparent or real circulatory emergencies and needs to have some basis for making quick decisions. One of the most important of these decisions is whether or not the situation is serious. A fainting spell may be quite benign or indicative of a very grave disorder. A sudden severe pain may be a simple cramp or a spasm or may be due to an embolus. An attack of suffocation at



night may be due to a spasm of bronchial asthma which will shortly leave the patient none the worse or it may be one of acute pulmonary edema. The purpose of the following discussion is to consider some of the more common cardiovascular upsets in which the patient or his family are apt to call the physician suddenly because they believe a serious emergency exists.

**Benign Syncope.**—One of the commonest sudden circulatory upsets is a fainting spell. Simple syncope is not very frequent in organic heart disease. There are many patients with valvular or myocardial disease who never faint and when a brief loss of consciousness occurs it is not always related to the organic lesion found in the heart. The only valvular disease in which syncope occurs with any frequency is aortic stenosis. This complication was discussed previously (Chapter 4). Occasionally the onset of any form of paroxysmal rapid heart action (Chapter 13) is associated with syncope. In most cases a simple fainting attack is a benign event occurring in otherwise healthy individuals. The precipitating factors for this are varied. The sight of blood, an overheated or crowded room, a sudden fright or the prick of a needle at the time of a vaccine injection or as blood is withdrawn for a Wassermann test, all may make some people faint. Another common cause is standing erect and fairly motionless for a considerable length of time. A sudden change of posture from the recumbent to the upright position is another cause of fainting. All these are intimately related to the nervous control of the vasomotor apparatus. In most of these instances loss of consciousness would not have occurred had the individual been recumbent. There is apt to be a splanchnic dilatation and a temporary cerebral anemia which naturally becomes aggravated if the body is in the upright position. For this reason it is customary to lower the head between the knees to revive one who has fainted.

If a person is seen during a faint it will be observed that he quickly grows pale and at the outset there is a fall in both the heart rate and in the blood pressure. Generally these early changes are not noted because by the time a physician arrives the heart rate has risen and the normal blood pressure level has been restored. There is much in this upset that resembles a vagal explosion or a temporary hypervagotonic state. In fact I recall once seeing a young man who could faint at will. After suddenly changing his position from recumbent to upright several times he would always faint. For an hour or so after a subcutaneous injection of 2.0 milligrams ( $\frac{1}{30}$  grain) of atropine sulfate, he could not be made to faint. It appeared that atropine paralyzed the vagus and prevented the reflex from taking place. This need not be the only or even the most common mechanism that controls benign syncope. The sympathetic nervous system may be involved in some reflex way that permits changes in blood pressure. From a therapeutic point of view it is wise to try tincture of belladonna, 10 drops after each meal or ephedrine sulfate 0.025 gram three times a day when such attacks recur with sufficient frequency to



merit medication. Wearing a tight abdominal corset may also be of some value. It is hardly necessary to recall that sudden massive internal hemorrhage needs to be thought of as a possible explanation of a fainting spell. When no organic disease is present, a patient having benign syncope should not be restricted in his activities and he should be assured that his heart is sound.

**Epilepsy.**—Obviously benign syncope of the type just described needs to be distinguished from petit or grand mal. The difficulty is not great if one witnesses the attack for there is no change in the pulse rate in epilepsy. I have made the error of confusing the two conditions when judgment had to be made from the history alone. The family history of epilepsy, the occurrence of an aura, the loss of sphincter control, the occurrence of convulsions and the postparoxysmal headache or sleep may help to differentiate the two. It is obvious that cardiac therapy would be of no avail in syncope due to epilepsy but that instead phenobarbital or dilantin would be used.

**Adams-Stokes Syncope.**—There is one condition in which syncope is a major and characteristic event and is the result of serious disease of the heart, *i.e.*, Adams-Stokes disease. Here sudden unexpected painless unconscious spells occur with or without convulsions. These attacks are due to a sudden failure of the ventricles to contract or a sudden marked slowing of their rate. The severity of the paroxysm will in a large measure depend on the duration of ventricular asystole. If this only lasts several seconds, the patient merely feels a "light wave" come over him. If it is longer he will lose consciousness and fall. If it lasts still longer convulsions occur and if the heart beat is not resumed after a few minutes, death ensues. When the attack ends the patient may feel perfectly well and be ready immediately to resume his former state. These attacks come suddenly, generally without warning or aura and with varying frequency. In some the spells are rare; in others there may be many during the same day, even producing a "status epilepticus."

The treatment for the syncopal attacks of Adams-Stokes disease, apart from whatever underlying condition may exist, is ephedrine or adrenalin. The former is given in doses of 0.025 to 0.05 gram ( $\frac{3}{8}$  to  $\frac{3}{4}$  grain) by mouth two or three times a day and the latter subcutaneously in doses of about 0.3 to 0.5 c.c. of 1:1000 solution. Propadrine may be substituted for ephedrine, the dose being the same. When the situation seems urgent and immediate effects must be obtained adrenalin should be used and repeated as often as seems necessary, even every two hours in some cases. When attacks recur at very rare intervals, it is hard to evaluate any therapy, but ephedrine may be tried by mouth. There are rare instances in which adrenalin has been injected directly into the heart during a prolonged period of asystole with recovery of the patient (Chapter 21, Fig. 62). It is obvious that under such circumstances a subcutaneous injection would be useless as blood flow has already ceased and no absorption would take place. There is also suggestive clinical and fairly



sound pharmacological evidence in support of the value of barium chloride (0.03 gram four times a day) in the prevention of recurrent attacks of this sort. Other measures that seem occasionally to be useful are inhalations of 1:100 adrenalin solution, full doses of atropine, metrazol intramuscularly or by mouth, thyroid extract and complete digitalization.

**Ventricular Fibrillation.**—Another type of syncope that results from a disturbance of the mechanism of the heart beat is one due to ventricular fibrillation. Although this is generally a fatal condition and very likely one of the causes of sudden death in patients with coronary artery disease, rarely it is a transient phenomenon. When it occurs the ventricles actually stop beating and fail to eject blood into the circulation. If contractions are not quickly resumed death results. This mechanism is apt to follow a preliminary period of ventricular tachycardia or frequent ventricular extrasystoles (Chapter 21, Fig. 61). When it can be established that syncopal attacks are due to such disturbances, quinidine sulfate (0.2 to 0.3 gram three times a day) should be tried as both from a theoretical and a practical point of view such medication may do away entirely with the attacks or at least diminish their frequency or severity.

**Carotid Sinus Syncope.**—Quite a different type of syncope that particularly has been studied in recent years is that which results from carotid sinus irritability. Soma Weiss and his co-workers discovered a considerable group of people with hypersensitive carotid sinus reflexes, some of whom are prone to fainting attacks. It has long been known that pressure over the carotid artery can often slow the heart beat and this mechanism has been used to stop attacks of paroxysmal tachycardia (Chapter 13) and to study certain cardiac arrhythmias, especially auricular flutter and heart block. It previously was thought that the method of action of this manipulation was by direct stimulation of the vagus nerve which lies beneath the carotid artery. It was shown, however, by Hering that this is not the correct explanation, but that the effect is produced by irritation of nerves surrounding the carotid artery at its bifurcation into the internal and external branches which produces reflex effects (carotid sinus reflex). Although under normal conditions the carotid sinus undoubtedly serves an important role in regulating certain bodily functions related to the blood pressure level, the heart rate, the effect of changes in posture, and other cerebral phenomena, in some individuals the sensitivity of this region is increased and results in a tendency to dizziness, syncope and convulsions. This occurs more frequently in patients with arteriosclerosis or hypertension but may be present without such changes. Increased sensitivity of the carotid sinus may also result from local irritation produced by a neighboring gland or a tumor in the neck. In some cases syncope is apt to occur as a result of a twist of the neck to one side or the other or to a peculiar position of the head from wearing a high collar.



Whenever a problem of syncope arises that is not readily explained by some other cause the patient should be tested for carotid sinus sensitivity. The proper part of the carotid artery should be sought for and will generally be identified by the finding of a local bulge or prominence at its bifurcation. First light and then more firm pressure and irritation should be applied, preferably with the patient sitting rather than recumbent. The pressure should be exerted for several to twenty seconds first on one side and then on the other. When a positive result is obtained the primary complaint of syncope will be reproduced and there is apt to result a fall of blood pressure or a slowing of the heart, although syncope may even take place without these effects. When the attacks of syncope can be ascribed to an increased sensitivity of the carotid sinus, it has been shown that the administration of daily doses of ephedrine sulfate (25 to 50 milligrams two to four times a day) may be effective in preventing such attacks. Occasionally atropine or tincture of belladonna (10 drops three times a day) may also inhibit the attacks. On the rare occasions in which a local tumor or gland is responsible for the attacks, removal of the cause is curative. Finally, Weiss and his collaborators have obtained permanent cures in a small number of cases by resecting the nerve plexus from the carotid artery.

**Cerebral Accidents.**—Sudden prolonged unconsciousness is commonly due to cerebral hemorrhage or thrombosis, subdural hematoma or cerebral embolism. With all these there is apt to be paralysis of one side of the body and if the speech centers are involved aphasia also results. It is important to realize that cerebral hemorrhage occurs in older persons with hypertension. When a sudden hemiplegia develops with or without unconsciousness in one who has not a significant hypertension there are several conditions to bear in mind. One must think of syphilis of the brain, polycythemia, Buerger's disease and brain tumor. When sudden paralysis occurs in younger people, especially when ocular muscles are involved, a rupture of a small intracranial aneurysm must be suspected. Such aneurysms are thought to be due to congenital deformation and not to syphilis. Finally embolism to the brain may cause sudden unconsciousness or hemiplegia. Some evidence of previous heart disease will then be discovered. It occurs mainly under four sets of circumstances—mitral stenosis, auricular fibrillation, cardiac infarction and bacterial endocarditis. When it is associated with the first two an embolus becomes dislodged from a sterile mural thrombus in the left auricle. When it develops from the third condition the embolus arises from a mural thrombus in the left ventricle that resulted from an infarction of the musculature following a coronary thrombus. Sterile mural thrombi may be present in the cardiac cavities for years without doing any harm and at present there is no way of detecting their presence or of predicting whether embolism will result. In bacterial endocarditis septic emboli are dislodged from the vegetations on the affected valves.

There is one aspect of cerebral embolism which has not been explored



but which may be of interest. It has impressed me that most patients with mitral stenosis complicated by cerebral emboli have little or no dyspnea. In general they have fairly good function and have been able to lie quite flat in bed. On the contrary, cerebral emboli are very rare in mitral cases with advanced cardiac failure or with orthopnea. One wonders whether or not the upright position of the neck and head makes it more difficult for the small thrombus, dislodged in the blood stream, to go up the carotid arteries. If the clot is heavier than blood it would remain in the lower portion of the stream and spare the cerebral circulation. Other possible explanations are that with advanced failure the velocity of blood flow is decreased and this would tend to diminish the likelihood of emboli, or that emboli are dislodged soon after mural thrombi develop. If the latter is true it would account for the occurrence of emboli early rather than late in the progress of mitral stenosis or auricular fibrillation. This problem needs further investigation.

It has been stated that cerebral hemorrhage occurs with hypertension. It is important to realize that the elevation in blood pressure is very apt to persist after the rupture of the cerebral vessel even if the patient is unconscious. Therefore, one should hesitate to regard the condition as due to cerebral hemorrhage if the blood pressure is normal. It may be found to be due to one of the other causes previously mentioned. At times one is thereby led to suspect the presence of a mural thrombus in the heart or cardiac infarction. Finally, sudden paralysis of one side of the body may take place only to disappear in a few hours. This may even be repeated. I have seen complete temporary hemiplegia recur several times during the course of a week in a man with hypertension. This very likely is due to transient spasm of the cerebral arteries and not to any permanent occlusion or rupture of a vessel. It is clear, therefore, that sudden cerebral accidents present a problem in differential diagnosis and that they should not be too readily regarded as instances of an ordinary apoplectic stroke.

The prognosis for patients having these cerebral accidents will naturally vary considerably. These hemorrhages are almost never instantly fatal. In this respect they differ from coronary attacks, which often kill instantly. When coma develops and continues for more than a day or so the outlook is grave. A slight fever and leukocytosis are common findings and are merely the result of the cerebral infarct or of the extravasated blood. A hypostatic bronchopneumonia is a frequent complication during the early days following a cerebral accident in elderly people and is apt to prove fatal. If the patient survives the first week he generally recovers. When paralysis has occurred some recovery of function is the rule and on the whole improvement in the power of the leg is greater than that of the arm.

The treatment for the condition is supportive. Occasionally lumbar puncture is performed or hypertonic salt solution or magnesium sulfate is given by mouth, by rectum or intravenously to diminish intracranial



pressure. The results from these methods of treatment have not been very impressive and the same may be said of phlebotomy. Caffeine sodium benzoate given intramuscularly in full doses (0.5 gram) may be useful as a respiratory stimulant and digitalis, if there are indications for its use such as auricular fibrillation or congestive heart failure, can be employed.

**Paroxysmal Dyspnea.**—A common cause for sudden alarm and one in which the physician may be hurriedly called is a paroxysm of dyspnea or palpitation. Paroxysmal dyspnea is most apt to occur at night and is often associated with Cheyne-Stokes breathing. It is common in hypertensive heart disease, in luetic aortic insufficiency and in disease of the coronary arteries. It occurs much less frequently in rheumatic valvular disease. The patient is awakened from sleep with suffocation and air hunger. He is agitated and develops a cough and a wheeze. The typical picture of acute pulmonary edema with abundant pink frothy sputum may follow. The patient often has a cold sweat and struggles for air. The attack when mild may last only fifteen minutes to a half hour and be followed by a fairly comfortable sleep. When it is more severe the dyspnea continues uncontrolled and the patient's life seems to be at stake. It is generally wise for the physician to administer a hypodermic injection of 0.010 to 0.015 gram ( $\frac{1}{6}$  to  $\frac{1}{4}$  grain) morphine and 0.6 milligram ( $\frac{1}{100}$  grain) atropine. Within fifteen minutes after such treatment most of the attacks subside. Occasionally a phlebotomy of 500 c.c. is extremely valuable. This is more beneficial if hypertension is present. The same result may be approximated by applying tourniquets to the extremities thereby producing a peripheral venous stasis. After the acute emergency is over adequate digitalis should be given to control future attacks.

Paroxysms of dyspnea occur in still other conditions and for other causes than those just described. Occasionally dyspnea and even acute pulmonary edema is the initial event in acute coronary thrombosis and treatment will then have to be directed accordingly. A patient may have a pulmonary embolism or infarct of the lung and be taken with sudden breathlessness and generalized pulmonary edema. It must be remembered that hemoptysis or bloody sputum frequently is absent in these conditions. Furthermore, a sudden increase in heart rate due to paroxysmal rapid heart action of one form or another may quickly produce air hunger, especially if it develops in one who already has significant organic cardiovascular disease. The differential diagnosis and treatment for these paroxysms have already been discussed (Chapter 13). Suffice it to say that such disturbances in the mechanism of the heart beat must be sought for because the proper treatment, which is generally very effective, will depend on accurate diagnosis. In the ordinary case of paroxysmal dyspnea (so-called "cardiac asthma") the heart rhythm remains normal and the rate only slightly accelerated. In these other conditions the rate is very rapid and the rhythm may be either regular or irregular. Some patients will require large doses of digitalis, others may be



readily controlled by pressure over the carotid sinus or the eyeball and a third group will require quinidine therapy, depending on whether the paroxysm is due to auricular fibrillation or flutter in the first instance, to auricular tachycardia in the second, or to ventricular tachycardia in the final instance.

**Pulmonary Embolism and Infarction.**—Sudden dyspnea or pain in the chest or both may be due to a pulmonary embolus. The condition may closely simulate an attack of coronary thrombosis. The clinical features of the latter have already been discussed (Chapter 6) and need not be repeated. A large pulmonary embolus may cause death within five or ten minutes. It rarely kills as instantly (in seconds or a minute) as does a coronary attack. Frequently the agony lasts longer and in many instances recovery occurs. Infarction of the lung need not take place during the early minutes or hours of such an attack as a longer time is required to produce these changes. The clinical features will vary considerably depending on the size of the pulmonary embolus and upon the suddenness of the occlusion. It must also be borne in mind that pulmonary thrombosis can occur because of local changes in the lung and is not always due to embolism.

The most alarming and sudden cases are due to emboli, dislodged from peripheral veins, especially those of the leg and the pelvis. Such venous thrombosis is common after surgical operations on the abdomen and embolism may occur whether there has been clinical evidence of phlebitis or not. These accidents are most common about ten days postoperative but may develop any time. Attention has recently been called to the fact that fatal pulmonary embolism may result from deep-seated phlebitis following apparently innocent traumatic injuries of the legs. In patients with organic heart disease pulmonary embolism follows the dislodgment of bits of mural thrombi from the right auricle, especially when mitral stenosis or persistent auricular fibrillation has been present. Local pulmonary thrombosis (not embolic) occasionally is found in patients with chronic passive congestion of the lungs and the changes thereby produced, although slower in development, are not unlike those caused by embolism.

Pulmonary embolism or thrombosis may cause sudden dyspnea with or without chest pain. When pain is present it does not have the radiation to the arms that characterizes coronary thrombosis. Cyanosis is also variable. The picture of shock is frequently present as in coronary thrombosis. If the patient survives the first twelve hours, fever and leukocytosis develop. When infarction of the lung results hemoptysis can occur. Bloody sputum, however, is not at all necessary to the diagnosis of pulmonary infarction. Jaundice is common with pulmonary infarction whether hepatic engorgement is present or not. The heart rate is apt to increase, the pulmonary second sound may be accentuated and the blood pressure may fall. Dilatation of the pulmonary artery occurs. This may be discovered by percussion, by x-ray examination or by a pulsation in



the second left interspace. At times a systolic murmur or a to-and-fro rub appears at the pulmonary area. Even changes in the ventricular complexes of the electrocardiograms resembling those seen in myocardial infarction may be found, *e.g.*,  $S_1$  is constantly present, slight depression of  $S-T_1$ ,  $T_2$  diphasic or slightly inverted,  $R-T_2$  not elevated and frequently depressed,  $R-T_3$  slightly elevated,  $Q_3$  often prominent,  $T_4$  upright or diphasic (Figs. 125, 126, 127). An absent R wave in Lead IV does not occur and this may serve to differentiate pulmonary embolism from coronary thrombosis. The differential diagnosis may be extremely difficult but a survey of all the data and particularly the search for a focus from which an embolus may have arisen and the relation of the attack to a previous operation, may enable one to arrive at the correct diagnosis.

**Coronary Thrombosis and Sudden Chest Pain.**—The lay public has now become quite aware of the significance of pain in the chest and its relation to coronary artery disease. This leads to many hurried calls for a physician. Many such emergencies turn out to be false alarms. Bearing in mind that coronary thrombosis is very common one must not forget that there are other causes of severe pain or distress in the chest. Some of the more important conditions that may simulate coronary thrombosis, such as pneumothorax and dissecting aneurysm, were discussed previously (Chapter 6). Besides these conditions, others and milder disorders at times turn out to be the cause of such attacks. I have been called to see numerous cases diagnosed as coronary thrombosis, in which later events showed that the whole disturbance was due to neurosis, herpes zoster, bronchial spasm, arthritis of the spine, acute pyelitis, acute hemorrhage from a peptic ulcer or other conditions. Great care, therefore, must be exercised in the interpretation of chest pain.

**Peripheral Emboli.**—Peripheral embolism furnishes further examples of sudden circulatory emergencies. These occur under the same circumstances as those mentioned above for cerebral embolism. The embolus may affect the kidney, spleen, intestine or limbs. When the abdominal viscera are involved the diagnosis may be quite difficult. More usual surgical conditions like acute appendicitis or ruptured peptic ulcer will need to be considered but occasional unnecessary operation will be inevitable. Splenic or renal embolism is best treated medically, while involvement of the mesenteric arteries when recognizable is apt to require an intestinal resection. A sudden severe pain in the arm or leg with blanching and coldness of the limb and disappearance of the arterial pulsation usually means an embolus. Many physicians still confuse arterial occlusion and venous phlebitis. In the latter condition the limb is not cold and there eventually develops swelling, whereas in the former there is no swelling and the temperature of the affected part falls. Some patients with embolism recover on symptomatic treatment. Embolectomy has been practiced at times with excellent results and at other times without success. It is best performed during the first six hours after the onset. The introduction of an apparatus which produces artificial



alternate suction and compression of the limb may be of aid in this problem and make embolectomy unnecessary. When gangrene has already developed in a limb, amputation will be necessary. Newer methods are being tried such as procaine hydrochloride injections in the lumbar sympathetic system and freezing the limb involved, but it is too early to judge their value.

**Paroxysmal Rapid Heart Action.**—Any of the forms of paroxysmal rapid heart action discussed in the previous chapter may be the cause of a cardiovascular emergency. When organic heart disease already is present, though compensation may be excellent, or even if the heart is structurally normal, a sudden change of rate from 80 to 160 or 200 may produce a state of collapse or shock or sudden acute pulmonary edema. At times with the onset of such a rapid rate actual syncope may take place, and in some cases anginal pain may occur. The physician should make every effort to examine the heart during the attack in order to establish the exact type of abnormal rhythm that is responsible, for the treatment (see Chapter 13) will vary with the different forms.

**Acute Hemorrhage.**—Many instances of acute hemorrhage have obvious causes and present an emergency situation. A massive nose-bleed, a large hemoptysis or hematemesis presents individual problems. Treatment is then directed at the underlying condition whether it is hypertension, pulmonary tuberculosis, cirrhosis of the liver or peptic ulcer. Occasionally with extensive internal bleeding diagnosis may become very difficult. Hypertensive patients may have acute hemothorax or hemoperitoneum. Of particular interest are cases of acute and massive gastro-intestinal bleeding. Because of the sudden shock and collapse these patients may resemble those with coronary thrombosis. Only in a day or so, when a dark or tarry stool is passed, may the true nature of the disease become apparent.

It can be readily seen from the foregoing discussion that the emergencies in cardiovascular disease are numerous. Sudden events and changes in the clinical condition of the patient are found to have a limited number of causes. They require a mechanism that can produce a sudden alteration in the blood supply of an individual part of the body or that suddenly changes the mechanism of the heart beat. A blood vessel can rupture or become partially or completely occluded. The heart may suddenly take on too rapid or too slow a beat as a result of a disturbance in the normal rhythm. Finally certain sudden nervous influences or reflexes may be set at work producing vasomotor changes or alterations in the blood pressure resulting in syncope. They all require individual analysis, for without this, intelligent treatment cannot be carried out.



## MEDICOLEGAL ASPECTS OF HEART DISEASE

PHYSICIANS have become more and more concerned with the relation of trauma to heart disease. The decisions that need to be made by courts, industrial accident boards and insurance companies often require medical opinions as to the existence of heart disease or the possible development or aggravation of pre-existing heart disease following trauma or accidents. We physicians have had little to do with the formulation of the existing laws and may believe that some are imperfect or even unwise. That is not our present concern. It is expected that we should offer intelligent and honest opinions as to the medical problems involved, leaving it to the judge and jury to interpret the law in the light of these facts and opinions.

As an illustration of a situation that seems unfair or at least unwise, the following may be mentioned. Two men were injured in the same way while walking and died several months following the accident, as a result of the injuries received. One was a young man twenty-five years of age who was perfectly well, and the other was a man sixty-eight years of age who was known to have had hypertension and angina pectoris. In the first instance the young man's life expectancy was shortened forty years or more, while in the second it was shortened possibly two or three years. In the eyes of the law in many courts or industrial accident boards the damages for the death of the two individuals would be identical. It would seem more just from a sociological point of view to differentiate the total amount of harm done to an individual by a specific accident according to the life expectancy, when a known organic condition exists. This and other aspects of the problem are making it difficult or impossible to obtain employment in industry for patients with heart disease, even when the cardiac abnormality is trivial.

There are many circumstances in which a physician is unable to answer questions of cause and effect if heart problems arise after accidents. The experience of most of us is confined mainly to the ordinary progress of heart disease. Our knowledge concerning the effects of trauma on the circulation is limited. Because of this lack of knowledge, the honest physician is often compelled to say he does not know whether a certain blow to the leg or chest or a fright can cause myocardial injury or any given arrhythmia. He may reason to the best of his ability and decide that the particular abnormality could not be or was a very unlikely result of the accident. He might state that the cardiac condition was a coincidence or was present before the accident, or was the result of the natural progress of disease. He would be influenced by his limited theoretical knowledge and by the fact that he had not associated such a causal rela-



tionship in his general practice. Similar points of view have been taken in the past concerning certain matters, only to find that years later such opinions were wrong. A good example of this is pain in the back following severe injuries. When such pain continued for months or years and physical examination including *x*-ray studies revealed no abnormality, the condition was often called neurosis or "railroad spine." In some cases it would even be inferred that the patients were malingering. No doubt many of these cases were functional or neurogenic but we know now that some were due to a dislocated and compressed intervertebral disc. Only in recent years has this condition been recognized.

The same situation exists in relation to heart disease. Formerly it was difficult to understand how a patient might develop angina pectoris or symptoms of coronary artery disease following trauma. If a man complained of anginal symptoms after an accident, I previously took the position that he must have had symptoms of this before but was not telling about them. The most that one could admit was that the condition was aggravated by the accident. Through the interest of Warburg in Denmark and Beck and Boas in this country, we have learned that direct blows to the chest, even without fractures of ribs or actual abrasion of the skin, can traumatize the heart muscle or coronary arteries so as to produce symptoms of coronary insufficiency. The "steering wheel" accidents that are so common now are good examples of the type in which contusion of the heart muscle occurs. There are now well-authenticated cases in which after an accident there developed definite angina pectoris or coronary thrombosis. Even instances of rupture of the heart or of a valve have been observed. These cardiac conditions not only may result from direct trauma of the chest, but also from severe injuries to other parts of the body or as a result of very severe and unaccustomed strain. I recall seeing an instance of a ruptured mitral valve which resulted from the severe strain of rowing a boat in a storm. The ruptured valve was found on postmortem examination. In this case increasing dyspnea and congestive failure developed, the man dying several months after the strain.

Arrhythmias of the heart may also result from unusual physical or mental strain. Many such cardiac irregularities produce no significant ill effects. Others are of more importance. I remember seeing a man who developed auricular fibrillation directly after falling down an elevator shaft. In another case, a man who had been operated upon for hyperthyroidism and had been symptomatically cured had a recurrence immediately after being held up in the street by a burglar. The symptoms of hyperthyroidism with auricular fibrillation promptly returned.

The most important point in many of these cases is the time relation between the accident and the cardiac abnormality. If the exact status of the patient before the accident is known, it is reasonable to assume that new objective or subjective evidence of disability occurring within minutes, hours or several days is due to the accident. Objective evidence



is obviously more reliable than subjective. It, therefore, is important to record findings such as heart rate and rhythm, blood pressure, electrocardiograms, the presence of râles in the lungs, x-rays, etc., as soon as possible after an accident. When the physician has to depend on symptoms, due regard must be had to functional and emotional reactions and to the possibility of malingering. However, the courts and some physicians have had a tendency to minimize the importance of post-traumatic neurosis. Many of the soldiers of the First World War who suffered severe neurocirculatory asthenia have ever since been much more handicapped than others who had fractures of bones or gunshot wounds of the abdomen or chest. In some of these cases the nervous symptoms have continued indefinitely, even when the question of war compensation did not enter into consideration. This has been found to be true in a follow-up study in England and has its counterpart in civil practice.

The following experience is illustrative. A man of forty who had always been well and strong had worked steadily for many years. He had been accustomed to manipulate very heavy steel beams. One day he was hit by a swinging beam and sustained a violent crushing blow to the body. An arm and ribs were fractured and it took a few months for physical recovery. When the patient became ambulatory he complained of weakness, palpitation, dyspnea on effort and tremulousness. These are the classical symptoms of neurocirculatory asthenia. I found no evidence of organic heart disease or any other significant organic disease. I regarded this working man as being in a worse condition than if he had recovered with a permanent limp in a leg or a contracture of an arm, but without any nervous symptoms. I felt certain that he would never be able to do the strenuous physical work to which he was formerly accustomed. I am afraid that such patients, when we feel that they are honest, receive poor treatment in medicolegal controversies.

The time relation is also very important when the question of aggravation of a pre-existing condition comes up. If a new chain of symptoms or findings develop a few hours or a few days after an accident and the clinical status before the accident appears to have been stationary, it is fair to assume that some degree of aggravation occurred. On the other hand, if examination reveals that there was no change for a few weeks and certain disabilities resulted thereafter, it is extremely unlikely that the accident was the cause of the change. In a case of this sort a very minor accident was supposed to have aggravated a pre-existing mitral stenosis and auricular fibrillation. I had reliable data before and after the accident which showed no change whatever. The patient claimed his breathing was worse, but I knew that the vital capacity of the lungs was in no way affected. This enabled me to maintain that the state of cardiac efficiency was not altered by the accident.

The main difficulty is in interpreting symptoms. When the problem of angina is involved, we are so dependent upon the subjective complaints that errors can easily be made, either for or against the interest of the



patient. We must first be certain of the diagnosis and then we should try to get some proof that the anginal pain is more frequent, more severe, of greater duration or brought on by less effort. If there is reason to doubt the veracity of the patient, he may need to be "shadowed" in order to check his statements. This does not come within the province of a physician as we are not detectives but rather guardians of the health of our patients. It may be the work of a lawyer or the insurance company. In all these matters scrupulous care and honesty on the part of the examining physician are paramount.

Finally, the troublesome question of "total and permanent disability" will be discussed. From a purely sociological point of view, this provision in insurance policies has often proved to be a great blunder. It has undermined the morale of many an honest citizen and has forced some physicians into practices which, from an ethical point of view, are at least open to question. Many of us would be happier if no such insurance policies had ever been written. Insurance companies are suffering tremendous losses which must be borne by all policyholders. This is partly due to the unexpected increase in chronic cardiovascular disease that has taken place during the past decades and partly to the economic depression of previous years. Added to this is the fact that many unrighteous claims have been granted because of the dishonesty of policyholders, occasionally with collusion of unscrupulous physicians.

The first difficulty comes in interpreting the term "total and permanent disability." Some totally blind or deaf people support themselves. A man could be bedridden and yet earn something reading proof or translating foreign medical publications, as I know one physician did for two years. It is the duty of the court to make a legal interpretation of this phrase. When a person takes out such insurance, he probably has in mind a protection against his inability to continue his accustomed work. If a man is an active obstetrician and has to climb stairs to get to his patients' homes, and must be able to exert himself vigorously when using forceps, then increasing breathlessness from cardiac weakness or from emphysema of the lungs renders him unfit to continue his occupation permanently. However, if he is a dermatologist with mainly an office practice, he may be able to carry on for some months or years longer. Now, are we to say that one or both are totally and permanently disabled? Many a disabled individual could carry on some mild or sedentary occupation, if he were trained differently and if such a position were available. But we cannot make a dermatologist out of an obstetrician overnight. Likewise a day laborer cannot easily be trained to be a cashier. The best we as physicians can do is to express an honest opinion as to whether or not a given occupation is an added hazard to the comfort or life of the patient concerned.

It is not our duty to help insurance companies undo losses that they have sustained. If we make an unwise investment in a home or in business, we have to suffer the loss. Insurance companies made what have



proved to be unwise contracts and must inevitably pay for their mistake. On the other hand, we must protect them against any false claims, by a most painstaking medical analysis, keeping in mind the interest of the patient and the company. Unfortunately, the final decision may depend on the amount of insurance involved. If a physician in general practice has insurance protection of \$4000 a year against total and permanent disability, his position may be different if he earns \$10,000 a year than if he earns only \$4000 yearly. When he develops angina pectoris and finds it hard to climb stairs or drive his motor car in wintry weather, he may well prefer to carry on because of a greater income, even if he may shorten his life or have more attacks of angina. If his income is no greater than his insurance, he is much less likely to want to suffer any more than necessary. A physician may quite reasonably urge one man to carry on and another to quit work when the physical disability is the same in both cases. At times it seems that the advantages of retiring are not great enough to warrant the financial loss involved. In some cases the joy and satisfaction of working compensates for the increase in discomforts. Some physicians would choose to have more attacks of angina but continue their practice. I believe that a man has a right to choose whether he will have more suffering and keep at work, or less suffering and accept his insurance protection, provided there is no doubt about the diagnosis and reality of the symptoms or disability.

There has been a move on the part of some insurance companies in very recent years that seems wise and should be extended. When a patient has coronary artery disease and his condition seems fair, it may be advisable to permit him to try to resume some work. If such an individual has been receiving total permanent disability benefits, some companies will give him a trial period of three months, during which time full compensation is continued while he resumes some or all of his duties. If at the end of that time it is clear that he cannot carry on, he again retires and continues to receive disability payments. If his progress is favorable after this three months' trial period, he then discontinues receiving the disability benefits and carries on with his work. This is a wise and useful provision, both for the insurance companies and the policyholders, for it may save the former expense and may help to rehabilitate the patient. Physicians should cooperate in this plan with the understanding that the insured will not jeopardize his interests by making an honest effort to return to work.

To return to the difficult problem of angina in which subjective complaints are so important, it may be necessary to perform special tests for diagnostic purposes. It also may be necessary to "shadow" the patient to find out whether he actually is unable to walk or work. It must be remembered, however, that detecting a man walking a mile or two in Florida in the winter does not necessarily mean that he can walk one block in New England. There are many individuals who can exert themselves a great deal in warm climates who can do very little in cold ones.



One should try to check the statements that are made in direct relation to the individual's ordinary working activities. Furthermore, it is fair for the physician to assume that his patient is honest, until he knows otherwise. This is particularly true when the family physician has known the patient for many years and has always found him to be honorable. I have known instances in which men were receiving insurance for total disability which I thought was entirely unjustified, and I have informed the companies to that effect. Unfortunately, too, I have seen instances when patients were disbelieved, had their insurance discontinued, only to prove their honesty by dying of a coronary thrombosis. The physician has a difficult task. Only by great care and wise and honest regard both for the interest of our patients and the protection of insurance companies, can the high standards of our profession be maintained and the services expected of us be rendered.



## 16

### THE SIGNIFICANCE OF BRONCHIAL AND OTHER FACTORS IN THE PRODUCTION OF DYSPNEA

BREATHLESSNESS is the most important symptom of heart disease. In the early stages of cardiac failure it may be the only evidence that the cardiovascular apparatus is inefficient. This is true whether the problem is one of valvular or non-valvular heart disease. The physical findings at this time may be entirely negative or may be very insignificant showing only those alterations which are commonly present in patients without any heart disease. It becomes very important, therefore, to be able to interpret intelligently the significance of this primary complaint—shortness of breath. There are other factors beside heart failure that play their respective roles in the production of dyspnea and some of these, especially the bronchial, deserve consideration.

Inasmuch as heart disease is extremely common and asthmatic bronchitis is also very prevalent it is not surprising that the two conditions are frequently present in the same individual. From a purely statistical point of view it would be expected that a considerable number of people might suffer from both diseases. In point of fact, however, I have seen so many patients with organic heart disease, especially rheumatic valvular disease, also suffering from asthma that I have been led to believe that the relationship is not merely coincidental. It may be that the prolonged pulmonary stasis of itself which accompanies heart failure in some way renders the bronchial tree more susceptible to the asthmatic state.



Another relationship which seems to be important is that other allergic stigmata such as a previous history of hay fever, urticaria or chronic eczema or a family history of such diseases are frequently detected in rheumatic cardiacs. One may suspect that such individuals are allergic in two different senses. In the first place they are sensitive to the proteins that bring about bronchial asthma, hay fever, urticaria, etc. In the second place they may be regarded as allergic in the sense that many manifestations of rheumatic fever are now looked upon as allergic reactions. These considerations may explain the undue frequency of asthmatic bronchitis in cardiac patients.

Bearing in mind that shortness of breath is the outstanding complaint in both conditions, it is most important to distinguish how much of the disability is due to each, for the prognosis and treatment will depend a great deal upon this distinction. This differentiation is obviously most important because although dyspnea due to asthmatic bronchitis is distressing, it comes and goes and is compatible with a long and fairly useful life, whereas when it is the result of cardiac failure, especially when it is of the paroxysmal type, the outlook is very grave. Furthermore, digitalis is helpful for cardiac dyspnea and useless for bronchial dyspnea and medication like ephedrine, adrenalin and potassium iodide which often is beneficial for bronchial dyspnea is practically of no avail in cardiac failure.

Such problems are best illustrated by actual practical experiences. Some years ago I was called to see a woman sixty years of age who was suffering from attacks of dyspnea, especially at night. She was the mother of a physician and had been seen by a most competent consultant who regarded the condition as hypertensive heart disease with paroxysmal nocturnal dyspnea or so-called "cardiac asthma." This diagnosis carries with it a very ominous prognosis for such patients do not live on the average more than about one or two years. I found that the dyspnea was only of several weeks' duration. Physical examination showed the customary amount of arteriosclerosis for a woman of her age. There was slight hypertension, the readings being 180 mm. systolic and 100 mm. diastolic. The heart was slightly enlarged, the action was regular with a rate of 90 and there was a slight systolic murmur at the apical and basal regions. There was no peripheral pitting edema and no hepatic or venous engorgement. So far, the findings were compatible with the diagnosis previously made, for many patients with paroxysmal cardiac dyspnea show very little of significance on examination, except cardiac enlargement. The signs in the lungs, however, gave the clue to the proper diagnosis. Râles were heard generally distributed throughout both chests and were readily audible over both sides anteriorly. They were inspiratory and expiratory and of the squeaking type such as are found in asthma or emphysema. There were no moist râles at the bases of the lungs. When râles are due to cardiac failure they are almost invariably moist and inspiratory and limited to the bases of the lungs. When they are generally



distributed as occurs in acute pulmonary edema they are also moist or bubbling, may occur in both phases of respiration, but the patient is then obviously in an acute serious state. Such was not the case here. The conclusion, therefore, was that the patient although elderly, hypertensive and somewhat arteriosclerotic, had no heart failure whatever and that the dyspnea was entirely due to asthmatic bronchitis. Subsequent developments confirmed this opinion. The digitalis she was taking was omitted and instead she was told to take 10 drops of the saturated solution of potassium iodide three times a day,  $\frac{3}{8}$  grain of ephedrine morning and night and to use steam inhalations. Either as a result of the medications or because of the changes in weather which often have an effect on an asthmatic state, she quickly improved and remained ambulatory for many years. Congestive heart failure never developed although she lived more than ten years after this experience, occasionally having recurring bouts of bronchial dyspnea.

The above experience illustrates the misinterpretation of the cause of dyspnea in a patient who proved to have no element of cardiac failure whatever. The following case illustrates the importance of differentiating the various factors involved when there is known organic heart disease. This patient was a man about forty years old who had been coming to the heart clinic for some years. He had rheumatic mitral stenosis and auricular fibrillation. There was a past history of hay fever. He had remained fairly well compensated taking 0.1 gram digitalis daily and was able to attend his little grocery shop. One day he was taken into the hospital and was found to be desperately sick. He had extreme dyspnea, the heart rate was accelerated and grossly irregular, there was a fever of  $101^{\circ}\text{F}$ . and he was irrational. Extreme dyspnea and orthopnea were the outstanding features. His condition was regarded sufficiently serious so that he was put on the dangerous list. What amazed the physicians in attendance and comforted the family was that I held out a fair hope for his recovery. I did so because a good bit of the respiratory distress could be accounted for on the basis of bronchial dyspnea. Although there were many moist congestive râles at the bases of the lungs, there were also numerous asthmatic squeaks throughout both lungs. He not only recovered but was able to continue at his light occupation for several years. I have had numerous other similar experiences in which the proper interpretation of the various factors producing dyspnea served as the main basis for offering a better prognosis than might otherwise have been made and in instituting more intelligent treatment.

The detection of a bronchial factor in cardiac patients is of considerable importance in treating ambulatory cardiacs in an out-patient clinic. Here we have a group of individuals belonging to the lower economic strata of life and if they are gainfully employed it is imperative, especially nowadays, to keep them so whenever possible. Picture a married man forty years of age who has mitral stenosis and auricular fibrillation, who works indoors as a salesman and is able to carry on fairly satisfactorily.



The occupation is not laborious and he manages to keep on the job and support himself. We all know that if such a man takes sick leave very often he will lose his job and that he may never be able to find another. He has been taking digitalis constantly and thereby the heart rate is kept fairly slow. Under these circumstances there will come a time when congestive failure will supervene, requiring a period of rest in bed. This will generally be ushered in by increasing dyspnea. On the other hand if the increase in dyspnea were due to asthmatic bronchitis we would not only be justified in permitting this man to keep at work but we probably would advise him to do so, in order that he might not jeopardize his earning power. Furthermore, increasing the dose of digitalis in these cases is generally of no avail, while a combination of ephedrine and potassium iodide often improves the condition. A familiarity with the bronchial factor in the production of dyspnea and the detection of the asthmatic type of râles have formed the background for the proper treatment of these patients. This has enabled patients to continue at work who would otherwise have been put to bed and has saved many of them their precious jobs.

There is a serious form of dyspnea, really bronchial in origin, which results from chronic emphysema of the lungs (chronic cor pulmonale). This generally is a slowly progressive process maturing into its severe stage later in life. Occasionally, however, it may appear in its advanced form at middle age. The chest is increased in its anteroposterior diameter, cyanosis develops early and may become quite marked and there is progressive dyspnea. The breath sounds are diminished in intensity, expiration is prolonged, cardiac dulness diminishes because of the overlying expanded lungs and the heart sounds become distant. The intercostal spaces are increased, the diaphragm is depressed and liver dulness is diminished. In a pure case there is right ventricular enlargement. Electrocardiograms often show right ventricular preponderance and complexes of low amplitude. An x-ray of the chest may show a prominent pulmonary conus. Objective signs of congestive heart failure, like pitting edema and enlarged liver, come late in the disease. There often is a marked reduction in the vital capacity of the lungs, because they are already fully distended and the enlarged chest cavity cannot expand much more. What is even more important is that the entire respiratory cycle lasts much longer than normal and it is difficult to increase the number of breaths per minute. This form of dyspnea and cardiac failure respond very poorly to digitalis but may be helped by ephedrine. The condition can be ameliorated by inhalations of oxygen, especially if administered with helium instead of nitrogen, or by a properly fitted abdominal belt. This belt can actually elevate the diaphragm and thereby aid its mechanical movements, increase the vital capacity of the lungs and improve the breathing. It is quite striking that in the early stages and often for many years patients with chronic emphysema may be quite short of breath on effort and yet have no orthopnea, being able to lie flat quite comfortably.



Closely related to this condition is one in which the primary pathological process is in the fine pulmonary arteries. It has been called Ayerza's disease, the patients with it, black cardiacs. Although it was first thought to be syphilitic in origin, most of those cases now recognized have been found to be non-luetic. The striking feature is the intense cyanosis with increasing severe dyspnea and weakness. This is a type of chronic cor pulmonale similar in its manifestations to the severe forms of "emphysema heart."

There are several other factors to be considered in interpreting breathlessness, *i.e.*, anemia, functional dyspnea and obesity. The first of these can quickly be dismissed. An appreciable degree of anemia is rarely of importance in patients who have cardiac failure from organic heart disease. Occasionally dyspnea is present solely as a result of anemia in patients whose cardiovascular apparatus is essentially normal. Moreover, appreciable dilatation of the heart may also be caused by anemia. Such patients may be erroneously treated for heart disease when liver or iron properly administered is all that is needed. It must be borne in mind that when the hemoglobin content of the blood is sufficiently reduced, dyspnea, especially on effort, can develop because of the diminished oxygen-carrying power of the blood. It is needless to say that digitalis cannot correct this defect. Furthermore, if the anemic state and dilatation of the heart persist for a long time, irreversible cardiac hypertrophy may take place.

A more important problem is dyspnea due to neurotic or functional factors. This frequently takes the form of "sighing breathing" and has been discussed in Chapter 12 and need not be gone into in detail here. Let it suffice to mention that it is common in those who have no organic circulatory disease and is not at all rare even in those with definite structural changes. When this condition is marked and maintained it may result in disturbing hyperventilation. Symptoms of tetany develop with tingling and numbness of the extremities. It is not difficult to interpret properly this type of dyspnea, especially if the physician observes the patients take these deep breaths (overventilation) when they complain they cannot get enough air. It has often been helpful in my experience to find that, after an individual has complained bitterly of not being able to breathe even while at rest, the vital capacity of the lungs proved to be normal. It is almost impossible for an individual to have distressing breathlessness from cardiac failure and yet have a normal vital capacity of the lungs. These considerations apply with equal force even when definite organic heart disease is present. Many patients with well-compensated valvular disease have dyspnea of neurogenic origin. The vital capacity of the lungs may be normal, the prognosis will be good and treatment must be directed at the functional state. Here also it is obvious that the proper interpretation of functional dyspnea will avoid errors in diagnosis, prognosis and treatment.

There is still another type of functional or neurogenic dyspnea that is



different from that called "sighing breathing." It takes on a variety of forms. I once saw a young girl who appeared well, had very few complaints and merely showed a very faint systolic murmur. The respiratory rate, however, was about 55 although there was no dyspnea and the mother was not aware of the rapid rate of breathing. In another case an hysterical Negro had a respiratory rate of 120 with otherwise normal findings. He had very few complaints and could lie flat. During sleep, however, the rate of respiration was normal. In two other cases there was marked dyspnea and orthopnea with very noisy and rapid breathing. One case was first regarded as due to serious heart disease, then to bronchial asthma. The patient was cured by psychotherapy. The other was a most extraordinary case featured by terrific attacks of suffocation. The patient even had an exploratory operation on the mediastinum in one hospital and a tracheotomy in another. He also seemed to be cured, at least temporarily, by psychotherapy. These may be regarded as instances of hysterical dyspnea.

Consideration must also be given to the weight of the patient who complains of breathlessness. Mention has been made of the fact that dyspnea may be the only early manifestation of cardiac failure. It is also well known that obese individuals frequently suffer from organic cardiovascular disease, especially hypertension. What is not fully appreciated is that obesity itself in an otherwise healthy individual can produce shortness of breath. The stout person cannot breathe as freely as the lean. The diaphragm does not descend as readily and when the adiposity particularly involves the abdominal region the diaphragm is apt to be held in a high position and the vital capacity of the lungs is diminished. It follows, therefore, that not all obese patients who have dyspnea have heart failure, and that even when there is definite evidence of organic heart disease, the dyspnea may be only partly, if at all, the result of weakening of the circulation. There are many obese patients whom I have seen who had been treated for heart disease when I felt convinced they were not suffering from anything more than obesity. Such patients will do better on a dietary regimen judiciously followed, directed at a slow reduction in weight, than they will on digitalis therapy. In fact, even if the obesity is permitted to continue they do well, as this form of dyspnea is not progressive and is apt to be elicited only on effort, particularly climbing of stairs. It is evident that the converse of this is true, *i.e.*, other things being equal, shortness of breath is more serious in a thin individual, for here despite the freedom of diaphragmatic movement there still is limitation in expansion of the lungs.

Because breathlessness is the primary evidence of heart failure, whenever this is a major complaint, either the patient or the doctor will quickly suspect the heart as the cause. It must not be forgotten that there are other causes of shortness of breath. Many diseases, particularly of the thoracic cavity, may produce dyspnea and occasionally they are overlooked when a weakened myocardium is thought to be present. Some of



these conditions are pneumonia, tumors of the lung, miliary carcinoma of the lung, Hodgkin's disease, aortic aneurysm, pulmonary tuberculosis, bronchiectasis, pneumothorax and septicemia. Under one set of circumstances or another the possibility of these diseases will need serious investigation, especially if the heart is found to be normal in size.

In this connection mention should be made of deformity of the chest as a cause of true myocardial insufficiency. There are occasional instances when the thoracic cage is so deformed as a result of early infantile paralysis or tuberculosis of the spine, or because of congenital and developmental deformities such as "trichterbrust" or "funnel chest," that the position of the heart is markedly distorted. Dilatation and hypertrophy of the cardiac chambers with congestive heart failure may result. This is produced by mechanical factors such as kinking or constriction of the large vessels leading to or from the heart, or by changes in the lung. Although this type of heart failure is quite rare it calls attention to the desirability of correcting as much as possible all chest deformities during the early years of life.

The foregoing discussion amply justifies the point of view that the degree of dyspnea needs careful appraisal. Various possible factors have different prognostic and therapeutic implications. Only in this way will serious practical errors be avoided.



## 17

### THE CLINICAL SIGNIFICANCE OF THE SYSTOLIC MURMUR

THERE has been a great deal of discussion concerning the causes and significance of a systolic murmur. Much speculation has been expended on the physical and mechanical factors involved in the production of murmurs and considerable effort has been made to correlate the presence of systolic murmurs with anatomical diagnoses. Despite this, much confusion remains especially in the clinical interpretation of such findings. Not so very long ago the detection of a systolic murmur meant heart disease and many an innocent, perfectly healthy person has been condemned as a chronic cardiac cripple, treated as such, restricted in his activities and made to live his life in the constant fear that so commonly characterizes the life of an organic cardiac. Others as a result of such a mistaken diagnosis would develop the fullblown picture of cardiac neurosis and thereafter live with the handicaps that accompany this condition. Some in defiance would lead a normal and active life for a great many years, to the amazement of the physician who originally



made the diagnosis and who either outwardly or inwardly gave a grave prognosis.

This point of view was quite prevalent before the First World War. The presence of a systolic murmur was often regarded as meaning mitral regurgitation and this diagnosis carried with it the inference that the mitral valve was diseased. During the war, however, there were so many young soldiers who had systolic murmurs and yet who were apparently well that much doubt was cast on its organic significance. The result of this experience was that the pendulum gradually moved to the diametrically opposite position. Whereas formerly all systolic murmurs were regarded as serious, a teaching developed that systolic murmurs had no clinical significance whatever. One authority went so far as to say "throw the stethoscope away," emphasizing the importance of eliciting the early symptoms of heart failure. Another expressed an extreme point of view that organic mitral insufficiency did not exist, that there was stenosis of the mitral valve or the valve was normal. This latter opinion was based primarily on postmortem experience, in which it was found that when the diagnosis of mitral regurgitation was made the valve was either found to be normal or stenosed. There is every reason, however, to believe that a middle course is much nearer the truth. Systolic murmurs cannot be entirely disregarded nor do they always mean heart disease. They deserve our most careful consideration, for only in this way will those of importance be distinguished from the insignificant ones. Furthermore, as will become evident in the following discussion, the proper interpretation of a systolic murmur may occasionally serve as the main clue to a diagnosis that otherwise will be entirely overlooked.

It must be conceded that on examining most normal people no murmurs will be heard over the precordium. Furthermore there is ample proof of the fact that disease of the mitral valve or a loss of the normal integrity of the valve leaflets can result in a systolic murmur. If the mitral valve of a dog is cut there will immediately develop a systolic murmur that previously was not present. The reverse of this, however, is not true for systolic murmurs can be present where the mitral valve is normal. Apart from other factors to be considered later, a systolic murmur best heard at the apex of the heart can be due to regurgitation of blood through the mitral valve whether the valve is structurally normal or diseased. If we have reason to believe that the valve is diseased the condition is regarded as "organic mitral insufficiency." If we believe that the valve is structurally normal the term used to designate the condition is "relative mitral insufficiency." This is a valid conception because there are numerous states in which for one reason or another the left ventricular cavity is enlarged or dilated and in this process of dilatation the mitral ring is sufficiently stretched so that normal leaflets can no longer completely close the orifice during ventricular systole and there results a regurgitation of blood. There are, therefore, two condi-



tions in which the mitral valve can be regarded as incompetent, one organic and the other functional.

Organic mitral insufficiency generally results from a previous rheumatic infection. Under these circumstances the valve is actually distorted, the free margins are apt to be thickened and retracted and although the slow process of stenosis has not as yet manifested itself, so that there still is free flow of blood during diastole from auricles to ventricles, there is a regurgitation through the incompetent valve during systole. It is often difficult to be certain whether or not a true organic mitral insufficiency exists because there need be no symptoms referable to the circulation and no other evidence of cardiac disease, not even hypertrophy of the heart. However, when a patient has had a previous history of rheumatic fever or chorea, and there is an apical systolic murmur of greater than slight intensity, especially if there is some cardiac hypertrophy and an accentuated pulmonary second sound, it is a fair presumption that the mitral valve is organically diseased and insufficient. It is no disproof of this contention to find a stenosis of the mitral valve on postmortem examination ten, twenty or thirty years later. When such a patient lives his span of life and dies of cardiac failure, mitral stenosis is very apt to be present. Fatalities from heart failure occur but rarely during the stage of mitral insufficiency so that ordinarily we have no opportunity to confirm the diagnosis at the autopsy table. There is one circumstance that does occur which enables us to see the mitral valve during the stage of organic insufficiency without stenosis, *i.e.*, subacute bacterial endocarditis. In this way a fatal disease develops in a patient who has only a systolic murmur long before congestive heart failure would otherwise have occurred and we are permitted to examine a mitral valve that is diseased but not stenotic. I have seen numerous such instances which have convinced me of the validity of making the diagnosis of organic mitral insufficiency.

There is another type of structural mitral insufficiency that is very difficult to diagnose which may result from calcification of the annulus fibrosus. This can readily be seen on fluoroscopic examination, but produces no characteristic physical findings. A moderate mitral systolic murmur is present in most but not in all cases. It generally occurs in older persons and may be present without any other significant evidence of disease or disability, although it also is found associated with organic disease of the mitral valve itself. This type of calcification of the mitral ring (in contrast to the leaflets) may or may not be associated with mitral regurgitation.

On rare occasions organic mitral insufficiency without stenosis does lead to progressive cardiac enlargement even with marked dilatation of the left auricle and congestive failure. It is not clear why most patients with simple mitral insufficiency do well for so many years and only a few develop heart failure rather rapidly. Possibly the degree of valvular



regurgitation, which is difficult to estimate during life and even post-mortem, is the determining factor.

Relative mitral insufficiency, on the other hand, is a common occurrence in a variety of conditions. When the heart is enlarged and the left ventricle is dilated in patients with hypertension, syphilitic aortic insufficiency or myocardial disease from any cause, a systolic murmur of varying intensity is frequently heard at the apex. There results a relative incompetency of the valve without any true progressive disease of its structure. It is of some importance to bear in mind the distinction between organic and relative insufficiency, for in the former we fear the eventual development of mitral stenosis or the subsequent complication of bacterial endocarditis, while with the latter neither of these conditions will develop no matter how long the patient lives.

Unfortunately many systolic murmurs cannot readily be classified in either one of the two groups just mentioned. It is this which has led to so much confusion and has given rise to such terms as accidental, cardio-respiratory, hemic or functional murmurs. It is not the intention to explain or analyze all these murmurs but rather to present some points of view that have developed from an extensive clinical study of the systolic murmur and which have proved useful in a practical way.

At the outset progress will be impeded if we do not start with clearly defined terms. By definition a systolic murmur, no matter how faint, must have duration; it must last an appreciable interval into systole between the first and second heart sounds. Because systolic murmurs have been so generally regarded as benign and inconsequential, medical students and young house officers have developed the habit of finding many systolic murmurs that never exist. They know that nothing will be said about it if some one else fails to hear it, especially as we know that faint murmurs may come and go, but they fear missing a murmur if it is present. The result has been a looseness and carelessness in examination and in terminology. Frequently I have failed to hear any murmur whatever on most careful auscultation when others have described a systolic murmur. One additional reason is that with the definition just given, a prolonged or impure first heart sound that frequently is heard, especially in thin-chested individuals with hyperactive hearts, will not be called a murmur for there is no true bruit extending into systole. With this definition in mind it is surprising how often classical instances of mitral stenosis will show no systolic murmur. The characteristic diastolic or presystolic rumble will be heard ending with a snapping first heart sound but no murmur will be heard during systole.

The other essential in this discussion is that we must indicate the intensity of the systolic murmur. Quantitative descriptions have become useful in describing other findings, *e.g.*, the amount of albumin or sugar in the urine, the degree of jaundice or peripheral edema. A very large trace of albumin, for instance, is not customarily seen in the simple albuminurias accompanying fevers, while it is suggestive of nephrosis;



contrariwise a slightest possible trace of albumin does occur with the former and not with the latter. Similar notations are applicable in describing systolic murmurs. For some years I have used the following terminology and although at first glance it may seem cumbersome, within a very short time those who have tried to use it have found it simple and reasonably accurate, so that different observers would coincide very closely in their decisions. Systolic murmurs are divided into six gradations. Grade one intensity is the faintest that is audible on the most careful auscultation. Although faint it must have an appreciable duration. This type of murmur is frequently overlooked if the examination is only casual. Grade six intensity is the loudest murmur that one ever hears. These murmurs are rare and are such that they can be heard with the stethoscope away from the chest wall. The other four gradations (two to five) fall in between these two extremes. They may be called I "very slight, II "slight, III "moderate, IV "loud, V "very loud" and VI "loudest possible" murmurs. With a short period of practice different observers will find that they generally will use the same notation and almost never vary more than one gradation in intensity.

With the foregoing as a background it was found that on examining over one thousand so-called "normal" or non-cardiac individuals, systolic murmurs of grade one intensity were fairly common, those called grade two were less frequent, and those of grade three quite rare. The interesting point was that in every instance in which a systolic murmur of grade three intensity was heard, although such patients were in the hospital primarily for some condition unrelated to the heart, such as prostatic disease, hernia, hemorrhoids, etc., definite evidence of organic heart disease was found. In fact this was also true of many patients that had grade two murmurs. There were several factors undoubtedly involved in the production of some of the murmurs of grade one and two intensity and in interpreting such murmurs these factors need to be carefully considered. These are fever, anemia, tachycardia, hypertension, hyperthyroidism and possibly nervous excitement. The last of these probably produces its effects primarily through its increase in rate. It does not follow that these conditions invariably produce systolic murmurs. There are other influences involved that are at present poorly understood which determine whether a murmur will result or not. However, it is fairly certain that they can be the specific cause of a murmur and therefore need to be carefully considered in estimating the significance of such murmurs, although they alone will not account for murmurs of grade three intensity or louder. It is obvious that when the above six conditions are known not to exist then the presence of a systolic murmur of more than grade one intensity is likely to be due to organic heart disease.

There is one possible mechanism in the production of a systolic murmur, which is common to some of the conditions, that I wish to discuss. This is the velocity of blood flow. The rate at which the blood flows



around in the circulation (not the heart rate) is increased in hyperthyroidism, anemia, fever and exercise. An increase in the basal metabolic rate of the body will be accompanied by a speeding up of the velocity of blood flow. It is interesting that many patients with these conditions also have systolic murmurs. I have frequently observed grade one or two systolic murmurs in patients with hyperthyroidism and found that they disappeared after the basal metabolism was brought to normal by subtotal thyroidectomy. Likewise, perfectly normal young men who show no murmur will almost invariably develop a grade one or two systolic murmur directly after a short brisk effort, such as running. These murmurs will also disappear as the heart quiets down. These murmurs have often been explained on the basis of temporary dilatation of the heart with relative insufficiency of one valve or relative stenosis of another. x-Ray examination of the heart in some of these conditions has failed to show any dilatation and in fact, at times, such as directly after a brisk effort, the heart seems to be a bit smaller than it is at rest. May it not be that the murmur is produced because the blood is ejected with a snap? It has been shown that there is a speed with which a fluid running through a tube does so without producing eddies and bruits and beyond which such disturbances do arise. This explanation seems logical when measurements of the velocity of blood flow show it to be accelerated. But even when this is normal, a systolic murmur can result from the same mechanism if we assume that the speed of ejection from the heart is accelerated for a short distance up through the aorta and pulmonary arteries, although the rate of flow through the entire circulation is unaltered. In any case there seems to be some relationship between the rate of ejection of blood from the heart and the development of a systolic murmur.

One by-product of this study was the finding of a transient systolic murmur developing almost invariably in normal persons after a brisk effort. This is of some importance, for it has become the habit of physicians who examine for insurance companies or civil service commissions to make such an exercise test and to draw certain inferences from the appearance of a systolic murmur. Although such a test can be of great value in uncovering mitral diastolic or presystolic murmurs that otherwise might be entirely overlooked, it is obvious that no significance whatever can be attached to the appearance of a systolic murmur after effort.

When a deliberate and careful attempt is made to interpret the significance of a systolic murmur, eliminating the factors discussed in the foregoing paragraphs, we are still left with a considerable number that are difficult to explain. Let us suppose that the heart is slow, there is no fever, hypertension, anemia or hyperthyroidism and a systolic murmur is present. If it is a grade three murmur, one is almost certain to find other evidence of organic disease such as cardiac enlargement, a systolic thrill, a diastolic murmur or significant electrocardiographic ab-



normalities. If it is of grade one intensity, unless other evidence of heart disease is present, no significance can be attached to it, although even some of these I believe eventually prove to be rheumatic or organic. If, however, the intensity of the murmur is grade two a most careful investigation must be made for a possible rheumatic background. Apart from the past history of rheumatic fever and chorea which will often be lacking, inquiry should be made of a family history of rheumatic fever or valvular disease and one should try to elicit a history of early nosebleeds, vomiting spells, undue nervousness or sweats or some unexplained illness in childhood. The point is that many rheumatic infections mask themselves in such obscure fashions and some do no more than produce a systolic murmur. In this way patients with grade two systolic murmurs will often be identified as having some form of rheumatic valvular disease. Others will be found to be suffering from some form of congenital heart disease such as patent ventricular septum, pulmonary stenosis, atrial septal defect, patent ductus arteriosus or coarctation of the aorta.

Having such indefinite evidence, it is not easy to convince oneself and much more difficult to convince others that a heart showing hardly anything more than a systolic murmur is not normal. This is particularly so because in some such cases, even those followed for a great many years, nothing further happens to incriminate the heart. This unfortunately does not occur in all cases. I have seen numerous instances in which patients with so-called "benign systolic murmurs" later developed conditions which proved that the original murmur, although in no way impairing the efficiency of the circulation, was due to an inherent structural defect. In some, a later bout of typical rheumatic fever serves to indicate that the early murmur probably was also rheumatic. In many the subsequent development of subacute bacterial endocarditis also proves that the original murmur was due to a minor rheumatic valvulitis or to some congenital defect because this complication is almost never superimposed on a previously normal heart. In still others as years go on, a systolic murmur that was regarded as functional or insignificant becomes readily identified as due to aortic stenosis, when a systolic thrill develops over the upper or midportion of the precordium. This has proved to be so in cases that I have followed even when the systolic murmur was originally more prominent at the apex than in the aortic area. In some the eventual appearance of the signs of mitral stenosis indicates that the early murmur was due to a mitral valvulitis. I have frequently followed patients who were well but showed a systolic murmur and found that ten years later a basal thrill and even x-ray evidence of calcification of the aortic valve developed. This is to be expected because long before a degree of stenosis of the valve has occurred sufficient to produce the classical signs, there must have been a time when the constriction was slight and the systolic murmur only faint.

A few illustrative experiences will help to emphasize how the proper



interpretation of an "insignificant systolic murmur" may lead to a correct diagnosis that would otherwise be overlooked. It was my duty some years ago to make routine examination of forty members of the first year class of the Harvard Medical School. There was only one who showed a systolic murmur. This was of grade two intensity. This young man felt well and did not consider himself sick. There was no previous history of rheumatic infection, no hypertension and the heart showed no other abnormalities. One might have dismissed this finding as of no importance and called it a benign or functional systolic murmur. On closer scrutiny it was found that his skin was somewhat moist and hyperemic and that there was a very slight tremor of the fingers. The heart sounds were also hyperactive. Although there was no exophthalmos or thyroid enlargement, hyperthyroidism was suspected, for which he was later treated as the basal metabolic rate was found to be +45 per cent. In this case the systolic murmur was the only feature that led to a diagnosis which otherwise would hardly have been suspected.

In the following instance (referred to in Chapter 10) attention to a simple systolic murmur also was the main clue to the diagnosis which had been entirely overlooked. A day laborer was sent into the hospital with a diagnosis of stone in the left kidney. A day or two before, he was taken with a sudden sharp pain in the left loin extending around the left part of the abdomen and down to the genitals. There was gross hematuria which was noted by the patient himself. He had been working when this occurred and complained of very little else. Examination was essentially negative except for a grade two apical systolic murmur. The urine showed gross and microscopic blood. The temperature and pulse rate were normal. The same diagnosis of left renal stone was made by the hospital staff, no attention being paid to the systolic murmur. When the patient was shown at a staff conference, in my attempt to explain the presence of the murmur, by direct questioning I elicited some additional information. Although he had been working steadily he had not been feeling quite as well during the previous few weeks. He had also known that he had some sort of a murmur for years but paid no attention to it as it never troubled him. On the basis of this I ventured the diagnosis of subacute bacterial endocarditis with an embolus to the left kidney. This suggestion was rather ill received but that afternoon, although the temperature was only 99.2° F., a blood culture was taken which was positive for streptococcus viridans. Cystoscopic examination which was contemplated was not performed and the progress was typical of subacute bacterial endocarditis. Postmortem examination performed some months later showed typical vegetations engrafted on an old rheumatic mitral endocarditis. This case also illustrates that during the years when the patient felt well and showed nothing more than a systolic murmur, the diagnosis of organic mitral insufficiency would have been justified.

In this final case the interpretation of a systolic murmur was helpful in deciding upon the proper treatment. A retired foreman, sixty years of



age, came under my care because of shortness of breath, especially at night. He was evidently suffering from congestive heart failure. The heart was enlarged, the rhythm was regular and there was a grade two basal systolic murmur. No thrills or diastolic murmur could be detected. There was no hypertension or rheumatic or luetic past history and there was no history of angina pectoris. The Wassermann reaction was negative. The question arose here whether or not we were dealing with a case of syphilitic aortitis. This deserved serious consideration for in about 15 to 25 per cent of such cases the Wassermann reaction is negative. Although I do not believe that disease of the wall of the aorta (so-called "roughening") is a factor in the production of basal systolic murmurs, this has been the current teaching and the systolic murmur might be explained in this way. x-Ray examination, however, showed definite calcified stenosis of the aortic valve. This finding not only made the anatomical diagnosis but served to rule out lues as an etiological factor, for syphilis never produces stenosis of valves. In this way the patient was spared a course of antiluetic treatment which not only would have been useless but might have been harmful. When his clinical condition improved as a result of ordinary treatment for congestive heart failure, a typical systolic thrill of aortic stenosis was felt and the murmur increased in intensity to grade three.

Finally, there are some general remarks about systolic murmurs that are not out of place. Too much emphasis has been placed on the transmission of murmurs in deciding whether they are "functional" or organic. The louder the murmur, the greater will be its transmission, and very faint murmurs are not transmitted. The location and the loudness are important, not the transmission. A loud aortic systolic murmur is transmitted to the neck and a similar murmur in the mitral area to the axilla because these locations are near the point of maximum intensity of these murmurs. A very loud murmur no matter of what origin will be heard over a large area. I question the importance of transmission of murmurs through the blood stream. (I have heard grade six murmurs transmitted down the arms and audible over the olecranon process of the elbow when they could not be heard over the brachial artery. They seemed to be transmitted well through bone) Furthermore, the distinction between organic and relative or functional murmurs often has erroneous connotations. The former sounds more serious than the latter, whereas relative dilatation of cardiac cavities resulting in functional systolic murmurs is apt to signify a graver condition of the heart muscle. There are some nervous persons who show hyperactive hearts and a systolic murmur without other evidence of organic disease who fall into a group of potential hypertensives. They often have a very slight fever, show flushing of the skin of the neck and as they are followed for years they eventually develop permanent essential hypertension.

It must not be inferred from the foregoing discussion that all systolic murmurs should be regarded with gravity. For the most part we should



do as we have been doing in the past. Patients with systolic murmurs and no other evidences of circulatory embarrassment may remain in good health for many years or even indefinitely. If there are no symptoms of cardiac weakness they should be allowed to do as they please. There really are no restrictions that they need. They may be allowed to enjoy the kind of physical activities and sports that produce no ill-effects. This advice is proper not because it is felt in all cases that the systolic murmur has no meaning, but rather because there is no advantage in enforcing rest or denying such individuals the ordinary pleasures of life. However, it must be clear that an attempt should be made to ascertain the cause of the murmur. As an aid in this direction proper terminology and estimation of the intensity of murmurs should be employed. In this way diagnosis will become more accurate, treatment in some cases will be directed more intelligently and vague terminology will become more clarified.



## 18

### THE PATIENT WITH HEART DISEASE AS A SURGICAL OR OBSTETRICAL RISK

THE physician is frequently asked by his surgical colleague whether a certain patient can stand an operation. This is by no means as simple a problem as it seems for it requires an answer to three different questions. First, the physician must help in the diagnosis of the condition and decide whether the problem is surgical after all. As will be seen there are many cardiac affections that present features which make them similar to and confused with acute surgical abdominal emergencies. Secondly, when there is an obvious disease that is amenable to surgical treatment one has to decide whether or not the prognosis of the cardiac condition is good enough to warrant subjecting that patient to a major operation. Finally, when an operation is contemplated the physician should have some idea as to the surgical or operative mortality in various cardiac disorders.

With regard to the first problem a physician often can spare a patient an unnecessary operation by making a correct diagnosis when the condition was supposed to be an acute surgical abdomen. In children, pericarditis or acute rheumatic fever may be accompanied by abdominal pain and tenderness, slight fever and leukocytosis and even nausea and vomiting. When joint pains are absent, acute appendicitis may be closely simulated. Inasmuch as there is no specific test for either condition, at times it will be necessary to perform an abdominal exploration to be sure of the diagnosis, for it is safer to find an occasional normal appendix than



to overlook appendicitis that develops into a fatal peritonitis. There will be occasions, however, in which the detection of some of the subsidiary evidences of rheumatic infection, such as epistaxis, a positive family or past history of rheumatic fever or rheumatic heart disease, will be sufficient to make one doubt the diagnosis of acute appendicitis and delay operation. After such a delay of twelve to twenty-four hours it may then become quite clear that there is nothing surgical about the condition.

On very rare occasions the sudden development of auricular fibrillation, in one suffering from some form of organic heart disease, may be attended by acute pain and tenderness in the epigastrium or right upper quadrant, nausea, vomiting, abdominal rigidity, slight fever, leukocytosis and slight icterus. The whole picture may resemble an acute cholecystitis. I have seen such a patient upon whom a cholecystectomy was performed following which there was satisfactory recovery. About one year later I saw the same patient go through a similar spell and she recovered on digitalis therapy. Reviewing the data of her previous experience it was clear that she had mitral stenosis and developed a paroxysm of auricular fibrillation which resulted in abdominal symptoms presumably due to an acutely engorged liver. After the gallbladder, which contained no stones, was removed the auricular fibrillation spontaneously disappeared and recovery was satisfactory. The second attack was like the first and was again accompanied by a sudden paroxysm of a rapid irregular heart action with an enlarged tender liver. This time the symptoms all subsided on appropriate treatment for the cardiac condition. It is obvious that the previous operation was unnecessary.

Another circulatory condition that produces symptoms resembling an acute surgical abdomen is embolism to one of the abdominal viscera. An embolus to the spleen or the kidney can result in sudden pain and tenderness in the abdomen with fever and leukocytosis. This is apt to occur in those cardiac patients who show evidence of mitral stenosis, auricular fibrillation or subacute bacterial endocarditis. The treatment is expectant and supportive and not surgical. When such an embolus involves the mesenteric vessels surgical treatment may be necessary. The diagnosis, however, is by no means simple and fortunately such a complication requiring operative resection is rare.

Finally, acute coronary thrombosis may resemble very closely an acute surgical condition of the upper abdomen. This has been adequately discussed in a previous chapter (Chapter 6). Suffice it in this connection to recall that all available methods may be necessary to make this differential diagnosis and to avoid the error of operating on a patient who is in the throes of a desperate cardiac affliction. The resemblance may be to acute gallstone colic, perforated peptic ulcer or acute pancreatitis. An electrocardiographic study may be invaluable and distinctive under these circumstances. Physicians have become aware of this diagnostic difficulty and now must be alert not to make the opposite error and overlook an abdominal disorder requiring immediate surgical intervention.



There are other acute circulatory emergencies that bring up the question of some surgical intervention in which the physician's judgment may aid in determining the proper treatment. At present, for example, it is a matter of careful judgment whether or not to try to remove a peripheral embolus to an extremity. Embolectomy is only of value the first six to twelve hours and it must be remembered that gangrene rarely develops in the arms. The use of suction apparatus seems to offer a fairly satisfactory method of treatment. This is really a peripheral pump and produces an alternating state of pressure and suction to the affected limb and thereby improves the blood flow through the part involved. Furthermore, novocaine injection of the sympathetic nerves to the limb involved may help to relieve the additional spasm that takes place with embolism, and freezing of the extremities is now being employed. It is clear, therefore, from the preceding discussion that when a physician is asked whether a patient can stand an operation, he should first try to decide whether there is any surgical problem involved at all or whether the cardiac disorder itself and its complication may adequately explain the difficulty.

The second point to determine is whether the life expectancy of the particular patient suffering from heart disease warrants undertaking the surgical procedure that is contemplated. Is it to be expected that the patient will live long enough to enjoy the results of the operation to make the temporary discomfort and risk worthwhile? This involves an estimation of the prognosis in the type of cardiac disease that is present, frequently a difficult matter. However, it is often fairly obvious that the patient cannot be expected to live more than a year or two and then one would hesitate in recommending an operation that is not urgent or for a condition that can be treated, even if less satisfactorily, by non-surgical methods. Too frequently women with hypertensive heart disease or mitral stenosis are subjected to pelvic operations, only to succumb to circulatory failure within a year or so after the operation. For many who survive the operations there must be some who make up the ordinary surgical mortality that attends such an operation. Patients with fibroid tumors of the uterus who also suffer from advanced heart disease with an accompanying poor life expectancy are much better treated by radiation than by surgery. Similarly, operations such as that for prolapse of the uterus or pelvic repair might well be avoided in such patients. Instead non-surgical methods may be employed. The same may be said for a simple hernia. In a word, whenever possible it is wise to employ simple non-surgical methods of treatment in patients with advanced heart disease although one should not hesitate to subject them to operations that are more urgent such as that for acute appendicitis.

The third consideration is an attempt to estimate the surgical mortality in patients suffering from different types of organic heart disease. Several years ago I made a review of 414 cases subjected to 494 operations. The surgical problems involved could all be regarded as major and



the cardiac abnormalities were all organic. In order to determine the role played by the heart in the outcome, deaths were divided into two types, "unexpected" and "inevitable." In the former group were included all those cases in which it seemed that had no operation been performed the patient would not have died. This, therefore, included all unexpected circulatory disasters such as coronary thrombosis, cerebral hemorrhage, embolism or circulatory failure. Even the ordinary complications frequently seen in non-cardiac cases, such as postoperative pneumonia, were regarded as "unexpected." Among the "inevitable" deaths were those due to the underlying disorder of the heart at about the same time as they might have died irrespective of the operation. This can be illustrated by the instance in which death occurred forty-eight hours after amputation of a gangrenous leg in a patient who was moribund from an acute coronary thrombosis with a femoral embolus. These "inevitable" deaths are of no importance in this discussion as they do not reflect the added risk that heart disease produces in withstanding operations. In this way it was found that the total operative mortality was 12.1 per cent and the "unexpected" mortality was only 6.3 per cent. This latter figure readily indicates that as a group patients with heart disease undergo surgical procedures fairly satisfactorily.

A more detailed analysis of the various types of heart disease disclosed some interesting relationships. There were only three "unexpected" deaths in 147 operations performed on patients with valvular disease; *i.e.*, a mortality of 2.1 per cent. In 167 operations on patients with non-valvular heart disease (hypertension, chronic myocarditis, etc.) there were eight "unexpected" deaths or a mortality of 4.9 per cent. Curiously enough the mortality among patients with permanent auricular fibrillation was also very low as there were only three "unexpected" deaths in 108 operations (3 per cent). Disease of the coronary arteries was found to increase the risk appreciably. In forty-one operations on patients with angina pectoris there were three deaths (7.7 per cent) and in twenty on patients with coronary thrombosis there were eight "unexpected" deaths (40 per cent). This latter figure was unduly high for it included some patients who were operated on in the midst of an acute coronary thrombosis either by mistake or when such an operation could readily have been delayed. There was one death in thirteen instances of syphilitic aortitis and none in six patients with paroxysmal tachycardia, three of whom had attacks during the operation. Congestive heart failure was found to add considerably to the operative risk as there were seven "unexpected" deaths in fifty cases (14 per cent). Dividing the patients into those with and those without nephritis showed that the mortality of the former was 14.8 per cent whereas in the latter it was only 4.9 per cent. Hypertension, on the other hand, was found to produce very little effect on the mortality. Patients with systolic readings over and under 160 mm. had mortality figures of 7.3 per cent and 5.9 per cent respectively.

It is clear that patients with organic heart disease who are well com-



compensated in general stand major operations satisfactorily. The risk increases if there is congestive heart failure and although at times it is necessary and advisable to operate in the presence of congestion, whenever it is possible to delay until a better state of compensation can be established, the operation should be postponed. The additional presence of nephritis adds to the surgical risk. The presence of angina pectoris likewise carries a somewhat greater hazard primarily because such persons are always subject to sudden coronary thrombosis or sudden death. The risk in cases with coronary thrombosis will not be as great as was indicated in this analysis if an accurate diagnosis of the cardiac condition is made and if operations are postponed until a sufficient time has elapsed after the attack of coronary thrombosis.

It is difficult to study the effect of different anesthetics in relation to surgical mortality in patients with heart disease. Individual clinics develop their own peculiar customs and preferences. In some ether is used a great deal, in others spinal or local anesthesia is favored and in others ethylene is a common choice. In the group discussed above various methods were employed (ether, local and intraspinal). There does not seem to be sufficient comparative data with regard to the relative merits of the various anesthetics to enable one to draw any conclusions. The choice must remain for the present an individual matter and rest upon the combined decision of the physician, the surgeon and the anesthetist. In general it may be said that when a local anesthetic will not suffice, ether is tolerated very well by patients with heart disease.

Finally a word must be said concerning the cause of death in these patients. They are naturally subject to the same hazards as a patient having a normal heart. Postoperative pulmonary complications are still the most common cause of surgical mortalities. Sepsis, phlebitis, hemorrhage, etc., will inevitably occur at times. The added difficulties that come because heart disease is present are the so-called "accidents" of heart disease. It seems that as a result of the operation, in some cases, emboli may become dislodged from silent mural auricular thrombi producing pulmonary infarction, hemiplegia or other arterial occlusions. Likewise occasionally an attack of coronary thrombosis may be precipitated by an operation. In this regard a marked fall in blood pressure in any patient having coronary artery disease must be avoided if possible because of the danger of precipitating coronary thrombosis. At times when an operation is performed for some septic process or when an infection results from an operation, subacute bacterial endocarditis may develop as a direct result of this infection. The ordinary type of congestive heart failure is only rarely precipitated by the operation unless infection or one of the above "accidents" has occurred. The added load or work which the heart is asked to perform as a result of the operation itself is no greater than the patient has already been demanding of that heart before the operation. As far as heart strength is concerned anyone who has been able to walk moderately without much discomfort is subjected to no



greater hardship in undergoing an operation. The "unexpected accidents" of heart disease, however, cannot be accurately predicted and they constitute the main factor in increasing the risk from surgery.

### HEART DISEASE AND OBSTETRICS

There is some similarity between the problems involved when a patient with heart disease becomes pregnant and when one is to undergo a surgical operation. There is one important difference, however, in that the former condition is to a great extent predictable and voluntary. This increases the responsibility of the physician for he will be asked whether pregnancy should be contemplated or after it has occurred whether it should continue. The intelligent answer to these questions will require not only a knowledge of diagnosis and prognosis of heart disease but also an insight into the social and economic life of the family involved.

There are several cardiac conditions that can readily be dismissed as having no influence on the question of pregnancy, for the response of the heart under these circumstances is practically the same as when the heart is perfectly normal. These conditions are all the forms of functional heart disease and well-compensated mitral insufficiency. Patients with benign irregularities of the heart or those showing insignificant faint systolic murmurs may be regarded as taking a normal risk. The same applies to those who have a louder mitral systolic murmur with or without slight cardiac hypertrophy in whom a past history of rheumatic fever or some other features indicate that there is an organic mitral regurgitation. Provided there has been no evidence of congestive failure and the patient's response to exercise is satisfactory, such patients can be regarded practically as undergoing a normal risk in pregnancy.

On the other hand there are some cardiac conditions which when present make pregnancy highly inadvisable. If a patient is suffering from subacute bacterial endocarditis it is obvious that the seriousness of the underlying disease does not warrant undertaking or continuing with pregnancy unless the infection responds to chemotherapy. If the mother is suffering from active rheumatic fever it is undesirable that she be pregnant at that time. Apart from any ill-effects that the pregnancy may have on the mother, and it is not certain that there are such ill-effects, the child may be born with a rheumatic carditis; for there are instances of acquired intra-uterine rheumatic heart disease.

The main problem arises in patients with mitral stenosis and aortic valvular disease. Of first importance is the state of compensation of the circulation. If there is any evidence of congestive heart failure or if such failure has once been present in the past, it is best to advise that no further pregnancies be undertaken. Even when an apparently satisfactory state of compensation can be established by appropriate medical treatment, the risk of recurrent heart failure is too great and the life expectancy of the mother is too short to make it advisable for such a woman to go through pregnancy. If it is undertaken, a high maternal



mortality must be expected. In general, women with rheumatic heart disease with congestive failure or with auricular fibrillation will have a maternal mortality of 50 per cent and only 50 per cent will have viable children. On the other hand, if there is no objective evidence of heart failure or dyspnea is either entirely absent or only of a slight degree, women with mitral stenosis, aortic stenosis or aortic insufficiency should be permitted to go through pregnancy. The only exception to this is the presence of permanent auricular fibrillation. This latter condition is generally associated with some signs of heart failure and for that reason alone will contraindicate pregnancy, but even when such has not been the case patients with auricular fibrillation are generally suffering from a disordered circulation that is too advanced to warrant taking the risk. Women manifesting those conditions that contraindicate pregnancy, *i.e.*, permanent auricular fibrillation, present or past congestive heart failure, should, therefore, be cautioned to avoid pregnancy and should be instructed in the methods of contraception.

There are differences in the advice which the physician should give when the question of the first or of subsequent pregnancies arises. What has been said above concerning well-compensated cases of aortic or mitral disease applies primarily to those women who contemplate their first pregnancy. Even when there is some doubt as to the exact state of the heart a slight added risk might be hazarded for the joy of having a child in contrast to a childless life. The situation is not quite the same if the patient already has one or more healthy children. Realizing that there is always a greater risk among pregnant organic cardiacs than among normal women, no matter how apparently trivial the disease may be, I feel that if there are already three children, under no circumstances should any more pregnancies be undertaken. It is not pertinent to the question to recall the instances in which women with mitral stenosis have satisfactorily borne eight or ten children. We see as patients only those who have survived. Many of the other multipara have succumbed, leaving their children motherless.

A distinction is made when there are already three children because that constitutes a satisfactory family both from a social and an individual point of view. It must be remembered that there is a strong familial factor in rheumatic heart disease and that with numerous pregnancies there is a great likelihood that one or more of the children will eventually suffer from rheumatic heart disease. If precautions are taken in families with stigmata of important hereditary nervous disorders like insanity, why should not similar considerations be given to this disabling form of heart disease? Furthermore, with numerous pregnancies among people of humble or modest means, the task is not ended with recovery from the confinement. The rearing of several infants and children without the aid of nurses and maids may prove to be a greater task than the pregnancy itself and more than the disabled heart of the mother can



stand. The social and economic status of the patient, therefore, deserves careful consideration.

When there is already one child and further pregnancies are contemplated the physician would do well to encourage a second pregnancy. Both the first and those subsequent pregnancies that are planned should take place during the early years of married life. Some women either through fear or improper medical advice delay this decision and lose valuable years thereby. Unless some acute episode has taken place, the cardiac condition does not generally improve with advancing years so that a pregnancy will be better borne earlier rather than later. Furthermore, the mother will enjoy the life of the children longer if they are borne during the early years of married life. A second pregnancy should be encouraged if there are no contraindications because a family is not secure with one child. Through some unfortunate accident or illness that child may die. Two healthy children, therefore, consolidate the family and also prevent the difficulties that often arise from a "spoiled" single child. When it comes to the third pregnancy one may rightly exercise a choice. The decision will often be arbitrary and rest upon the desires of the parents.

The foregoing considerations apply to those women who are not pregnant and come to the physician for advice. The problem is somewhat different if the patient is already pregnant. At the outset it must be emphasized that every woman with organic heart disease should be cautioned to consult her physician at the first suspicion or indication of pregnancy, so that a decision may be made early during pregnancy. This is particularly important in public cardiac clinics when patients are seen only at infrequent intervals. If a menstrual period is missed it should quickly be determined whether pregnancy has occurred. This can be done quite satisfactorily by means of the Aschheim-Zondek test. Once it is known that the patient is pregnant a decision should be made whether or not abortion should be advised. If there is or has been definite congestive heart failure or if permanent auricular fibrillation is present, termination of pregnancy is indicated. The procedure involved is comparatively harmless during the first few months and, therefore, the decision should be made early. If it is thought advisable that abortion should not be induced the patient should be advised concerning her activities and should be under frequent medical observation. It rarely is necessary in these cases to administer digitalis unless there is congestive failure or permanent auricular fibrillation. Women should be cautioned against overdoing. Frequent shopping trips may precipitate symptoms of congestive heart failure. There still prevails in the minds of many women and some physicians that an expectant mother must "harden" herself by deliberate exercise for the trial of labor. I have seen such a patient systematically walk three miles a day over hilly ground at the advice of her obstetrician, month after month, until she developed heart failure.



A respiratory infection is also a frequent cause of upsetting the state of compensation and in so far as possible it should be avoided. When a minor respiratory infection has occurred more than the usual care and rest in bed should be advised.

The earliest evidence of weakening of the circulation will be a slight increase in the dyspnea, a slight non-productive cough and the presence of a few râles at the bases of the lungs. Inasmuch as some subjective dyspnea may have been present before the pregnancy occurred, and many women without heart disease may complain of slight dyspnea during pregnancy, it is no simple matter to appraise this symptom. It has proved very helpful to me in following these patients to watch the vital capacity of the lungs. When the patient progresses favorably it will be found that the vital capacity of the lungs, which is an excellent objective index of the degree of breathlessness, will remain essentially unchanged throughout pregnancy. It is surprising that even at the eighth month when the abdomen is markedly distended by the enlarged uterus the movements of the diaphragm are not impeded sufficiently to lower the vital capacity. By this simple determination one can obtain a check on the patient's clinical condition. Obviously the physician should carefully examine the patient during these frequent visits, watching particularly for basal râles apart from observing the blood pressure and urinary findings. Pitting edema of the legs which is such an important sign of congestive heart failure is not trustworthy during pregnancies because many women manifest this as a result of pressure of the uterus on the pelvic veins or for other reasons.

If any definite evidence of congestive failure is detected the patient should be kept in bed and treated appropriately. If this occurs during the first three months and there is already one healthy child, abortion should be advised after the state of compensation has been improved, even if what appears to be satisfactory recovery of the heart has taken place. If heart failure occurs in the latter part of pregnancy, the decision is difficult. One needs to consider whether the child is viable or whether the pregnancy can be carried along far enough to obtain a viable child. In some cases abortion will have to be carried out at the fifth, sixth or seventh month with the hope of saving the mother. It is readily seen that there will be occasions when decisions are difficult and when the outlook is not very promising whatever course is taken. However, once definite evidence of congestive failure has developed, that patient should be confined practically to bed for the remainder of the pregnancy.

There is one type of heart failure that occasionally develops rather suddenly in pregnant women even when the heart was normal before pregnancy. This may take the form of acute pulmonary edema during a toxemia of pregnancy with a rising blood pressure. The mechanism is an acute left ventricular failure. This condition is amenable to cardiac therapy. Complete recovery can take place following the use of mor-



phine, digitalis and phlebotomy when necessary. In such cases the heart may return to normal and pregnancy may continue in the usual manner.

There are sharp differences of opinion concerning the choice of operative procedure in pregnant women with heart disease. Some obstetricians believe that a cesarean operation causes the least disturbance to a damaged heart. Others maintain that it is best to permit the patient to give birth to the child in the normal way. There probably is no single rule that will be applicable in all cases. If it is expected that the labor will be short and easy and the state of cardiac compensation has been satisfactory, I believe a cesarean section should be avoided. When, however, it has already been decided to perform sterilization, a cesarean operation should be done and the tubes should be tied off at the same time. One should hesitate to do this at the time of the first pregnancy, as during the operation it is not known whether the child is viable or in normal health. I recall an instance in which sterilization was performed at the time of the first confinement and the child died during the first twenty-four hours. The mother recovered satisfactorily and remained a well-compensated cardiac. She probably would have been able to go through one or two more pregnancies, but of course the sterilization prevented conception. Sterilization should, therefore, be advised when it is certain that under no circumstances will the mother be allowed to go through any further pregnancies. In my own experience it has seemed that pregnant cardiacs do very well with ether anesthesia and tolerate an abdominal section with very little difficulty. When heart disease is advanced and the state of the circulation is somewhat in doubt this method disturbs them less than a delivery from below. The intelligent cooperation between the obstetrician and the cardiologist has brought about a tremendous fall in the maternal mortality of those suffering from heart disease. This has been well exemplified in the special cardiac clinics established in lying-in hospitals, such as that instituted by Hamilton in Boston. We now are able to advise patients whether or not to undertake the hazards of pregnancy and then to guide them along to a successful termination with a surprisingly small risk.

When the above general rules are applied the maternal mortality will be less than 3 per cent. This is still ten times as great as that in normal women. There will be unexpected instances of congestive failure in some cases of mitral stenosis, or fatalities may occur from gross emboli or bacterial endocarditis. I suspect that the risk can be a good deal less than the mentioned 3 per cent if meticulous care is taken. In my own experience there has been no mortality whatever except among a few patients who became pregnant against my advice.

Finally, one may ask whether the life span of cardiacs that recover is shortened by pregnancy. This is a difficult question to answer. Some statistics indicate that women with rheumatic heart disease (not dying in pregnancy) are five years younger at the time of death than women



with comparable conditions who have not been pregnant. This difference may be due to the fact that some develop congestive failure, recover partly and succumb a short time after delivery. If heart failure does not develop it seems unlikely that life has been appreciably shortened.



## 19

### FACTORS CONCERNING PROGNOSIS IN HEART DISEASE

ALL physicians at one time or another have had the disturbing experience of making an absolutely wrong prognosis while treating patients with heart disease. This occurs even when the diagnosis is correct. Such wrong prognoses are made both when the condition seems hopeless and the patient recovers, and contrariwise when the patient has been doing satisfactorily and dies unexpectedly. It is particularly humiliating to the individual physician and harmful to the general medical profession when a hopeless prognosis is given and the attending physician is dismissed, if one of the irregular practitioners is called in and, in the natural course of events, the patient recovers. Such an experience makes an everlasting impression on the parties involved and on their friends and serves to place in disrepute the entire medical profession. It is not surprising that the lay public should feel as they do under these circumstances. Mistakes in prognosis, more so than mistakes in treatment, are responsible for the growing activities and importance of the numerous unorthodox medical cults that prevail today. The purpose of this discussion is to throw some light on the factors in the prognosis of heart disease so that such errors may be minimized.

There are two parts to the problem of prognosis that frequently arise in cardiac patients. The first concerns itself with the immediate and the second with the ultimate outcome. The factors that concern the latter are very involved and difficult to measure. It is almost futile to speculate as to exactly how long a patient with compensated aortic or mitral valvular disease will live. There are so many uncontrolled and unpredictable influences at work that although one can formulate some average figures that roughly guide our concepts of life expectancy it is difficult to apply them to individual cases. Two patients start out at the age of fifteen years with apparently similar lesions, *e.g.*, a slight aortic or mitral insufficiency. One carries on in good health to the age of fifty or sixty and the other picks up a minor infection that initiates subacute bacterial endocarditis which proves fatal when the patient is thirty years of age. The same marked differences in ultimate outcome characterize



disease of the coronary arteries. One patient with angina pectoris has a sudden exitus shortly after the first symptoms develop and another continues in fairly good health for a great many years. When we realize that there may be such extreme variation in life expectancy among apparently similar cases, for the present it seems idle to try to predict exactly how long many of these patients will live. Perhaps it is just as well that prognosis is no more accurate. It enables both the physician and the patient to be hopeful and not to feel that the day of judgment is absolutely fixed.

The difference between predicting immediate and ultimate results is well illustrated by the influence of the presence of auricular fibrillation with valvular disease. Whereas auricular fibrillation indicates a more advanced state of rheumatic heart disease and on the average the life expectancy is shorter in the rheumatic cardiacs with auricular fibrillation than in those with regular rhythm, the immediate prognosis and expectations for improvement are better if this irregularity is found than when the rhythm is normal and the same degree of cardiac failure is present.

It is obvious that in any form of heart disease, other things being equal, the more ill the patient seems to be, the more dyspnea, the more peripheral congestion, and the like, the more serious will be the outlook. On the other hand, there are many patients with marked symptoms and physical signs of circulatory embarrassment in whom the prognosis is a good deal better than in others with much milder evidences of the same type. This occurs because other factors less obvious are not the same in the two groups of cases. We must try to explain why some patients with heart disease unexpectedly do better and others do worse than predicted, and even why, in some cases that are considered entirely hopeless, recovery takes place.

One simple aid in estimating prognosis is the heart rate at which the patient develops congestive failure. If two cases of mitral stenosis and auricular fibrillation present the same degree of cardiac failure, but one has a heart rate of 130 and the other a rate of 80, in general it may be said that the prognosis is better in the one with the rapid rate. Here it is evident that digitalis and other treatment may be expected to slow the rapid rate and effect a good deal more improvement than when the rate is already slow. In other words, when the heart rate is slow and there is considerable peripheral edema, not so much improvement can be expected as when the rate is rapid. This is especially true when the disturbance is one in which it may be predicted that slowing will be obtained such as is the case in auricular fibrillation.

The size of the heart is of some importance in judging prognosis. The larger the heart, other things being equal, the poorer the prognosis. This does not mean that one patient with a large heart may not do better than another of a different type with a small heart. I recall seeing a patient with mitral stenosis and auricular fibrillation who had marked congestive heart failure. She improved strikingly on ordinary treatment



so that all evidence of congestion disappeared and in fact the vital capacity of the lungs, which had been very low on admission, rose to a normal level before leaving the hospital. She walked up two flights of stairs with one of the physicians and showed, if anything, less respiratory distress than he did as a result of this effort. One might have predicted that she would do extremely well. Her heart, however, was very large, almost filling the chest, and she died within a year. Such experiences are not uncommon.

One of the most important factors that needs proper appraisal is the role that infection plays in producing symptoms and signs of heart disease. It is evident that infection, although violent, may come to an end. A patient may be desperately ill with respiratory distress, very rapid heart, even showing signs of generalized congestive heart failure, and yet recover so that all evidence of circulatory embarrassment disappears. Such a patient may not only feel well and remain well indefinitely, but physical signs of heart involvement, such as murmurs, may disappear entirely. This sort of dramatic improvement is not altogether uncommon in young people. The reason that this can occur is because under such circumstances almost the entire picture that develops is due to an infection, generally rheumatic fever. This is apt to be accompanied by a pancarditis with involvement of the pericardium, myocardium and endocardium. This sort of an infection may not only terminate, but recovery from it can be practically complete. I have seen such patients who were ill enough to be on the danger lists in hospitals but who recovered entirely and showed no signs or symptoms of heart disease in subsequent years. If, on the other hand, the same degree of circulatory failure occurred without an infection merely as an end-result of a burnt-out, long-standing, chronic cardiac condition, recovery of the type described would be extremely rare. Improvement may occur, so that the patient may become ambulatory but there will always remain a varying degree of disability which is generally considerable. The inference to be drawn is that when infection of the heart is present, although the condition may be very critical, and frequently fatal, one has reason to be hopeful, for a satisfactory recovery is always possible.

The role of bronchial dyspnea in heart disease needs special consideration in judging prognosis. There are a great many people with bronchitis of the asthmatic type and emphysema. There are also a great many individuals who have one form or another of heart disease. It would be quite natural, therefore, to find a certain number who happen to have both conditions. Furthermore, dyspnea on effort or at rest and paroxysmal dyspnea are symptoms that are common to both heart disease and asthma. It, therefore, becomes very important at times to determine how much of the respiratory distress is circulatory and how much is bronchial in origin. The importance of the differentiation lies in the fact that the prognosis in a patient with marked dyspnea of bronchial origin may be excellent whereas if the dyspnea were mainly cardiac, the outlook



would be quite grave. There are numerous instances of mistaken prognoses because of the improper interpretation of the mechanism of dyspnea. For cases illustrating this problem and for the details that help in avoiding such errors see Chapter 16.

There are other influences at work in the production of dyspnea that have no direct bearing on heart disease which one must clearly distinguish, because in so far as they are present the prognosis is so much the better. Obesity in itself can produce shortness of breath when the heart is normal and, therefore, can be responsible for a part of the dyspnea when there is heart disease. The same degree of dyspnea will have less significance in an obese than in a thin individual if the other findings are the same. In fact, there are many obese patients in whom a diagnosis of heart disease is made because they complain of shortness of breath, who really have no structural disease of the heart at all. Likewise, patients with nervous weakness and debility complain of shortness of breath and palpitation and often are regarded as having organic heart disease when the entire problem is functional. Furthermore, there are many patients with compensated organic heart disease whose symptoms are in the main functional or neurotic in nature. In so far as that is true the prognosis is so much better. There is one particular aspect of this question that deserves attention. I refer to a group of comparatively young individuals who complain of shortness of breath even while at rest. They are apt to say that they cannot get enough air and will frequently be seen to take deep breaths ("sighing breathing"). They actually overventilate their lungs and still want more air, in contrast to patients with "cardiac asthma" who really cannot take a deep breath and who have true respiratory distress. They will show no signs of cardiac failure and no evidence of serious organic disease and yet feel short of breath without effort. When spontaneous paroxysmal dyspnea is of importance it will generally be accompanied by obvious signs of grave heart disease, whereas in this type of "sighing breathing," examination will reveal no essential abnormality. The distinction between these two types of dyspnea occurring when the subjects are at rest is most important for on the one hand the prognosis is excellent and on the other it is very grave.

It is obvious that the facility with which the symptoms of heart failure (dyspnea, edema and chest pain) develop and with which they disappear under treatment is of some help in judging prognosis. Edema of the legs that develops only at the end of the day and disappears overnight is less serious than edema that persists all day. The same is thought to be true of anginal pain coming in some patients only if they hurry up a hill and in others even when they try to shave themselves. Similarly, if the patient improves quickly, one may regard the outlook as better than if it takes many weeks to accomplish the same result. Although there are exceptions to the foregoing principles, in general they will be found to be true.



In following a case of mitral stenosis I have found that the development of hypertension is indicative of a better prognosis than one might otherwise expect. As a group, the majority of patients with mitral stenosis will have died before they reach the age of fifty. More recently we have learned that an appreciable number will live to sixty and some to more than seventy. It is curious that hypertension is much more common in this older group of patients with mitral stenosis than in the general population for corresponding ages, despite the fact that mitral stenosis of itself in younger people is accompanied by a blood pressure somewhat lower than the average. Why this should be so is not altogether clear, although it may be that the original rheumatic infection that produced the mitral stenosis also had some insidious effect on the blood vessels causing the hypertension. In any event, I have frequently been aided in giving a better prognosis than would otherwise be warranted by finding the blood pressure elevated. For example, the presence of a systolic blood pressure of 140 to 160 during some acute cardiac emergency like an infarct of the lungs with generalized pulmonary edema in a patient with mitral stenosis would lead one to hope for recovery although the patient may appear desperately ill. Similarly, if the blood pressure gradually rises to 160 systolic or more from year to year, a patient with mitral stenosis may progress more favorably than one would otherwise expect. The development of moderate hypertension, therefore, is a favorable sign during the course of mitral stenosis.

It is well to contrast the prognosis of dyspnea and congestive heart failure in cases of mitral disease with that in aortic valvular disease. Many patients have well-marked heart failure with mitral stenosis, recover compensation and again become ambulatory. They may carry on for many years going through repeated breaks in compensation. On the other hand when a patient with aortic stenosis or insufficiency, rheumatic or luetic, once has dyspnea and peripheral edema his months are often numbered. However, the aortic patient is apt to be stronger and suffer less from dyspnea than the patient with mitral stenosis, until this final break in compensation does occur. The effect of tricuspid stenosis on prognosis was discussed in Chapter 4. It is of interest that when this lesion is present the length of life is much greater after heart failure develops than in any other form of valvular disease.

Of special significance in considering the question of shortness of breath is nocturnal paroxysmal dyspnea. This occurs most frequently in patients with hypertensive, luetic or coronary artery heart disease. This symptom even more so than pulsus alternans, gallop rhythm, and bundle branch block carries with it a very poor prognosis. Most patients will not survive the first development of nocturnal dyspnea more than a year. There are numerous patients, however, whom I have seen with pulsus alternans and gallop rhythm who did well for more than five years. We must try to individualize in our prognosis and it will be found that if some acute episode, which can disappear, brought to light these grave



signs they need not have the same hopeless outlook. Many patients will show Cheyne-Stokes breathing, pulsus alternans and gallop rhythm during the height of a coronary thrombosis or an attack of paroxysmal tachycardia and yet recover and do well for years. In fact, in some of these cases of paroxysmal tachycardia the sign of ill-omen disappears permanently and the patient remains well indefinitely. In general, however, it is true that when nocturnal dyspnea, Cheyne-Stokes breathing, pulsus alternans, gallop rhythm or block of one of the branches of the bundle of His occurs, the prognosis must be guarded.

Thyroid heart disease is unique in that the prognosis almost invariably is much better than it would be with the same apparent evidence of cardiac embarrassment if there were no hyperthyroidism. When the diagnosis of hyperthyroidism and heart disease is properly made and effective treatment is instituted, patients who otherwise could rightly be expected to be incurable and even helpless invalids can generally be restored to comparative comfort or complete health. There is no other condition in which such extraordinary improvement occurs and is so well maintained as in hyperthyroid heart disease. For this reason the diagnosis is most important, although it is still too frequently overlooked. It must be said, however, that the apparently sick patients are those who generally, but not invariably, have an additional and independent form of heart disease as well, such as mitral stenosis, hypertension or coronary artery disease and that even here the prognosis is still very good.

In contrast to hyperthyroidism, the presence of chronic nephritis in a patient with heart disease makes the prognosis much worse. Quite often when the term "cardiorenal" is used in describing the clinical condition, there really is no significant nephritis. The urinary findings are those of passive congestion and not of true renal insufficiency. It is quite important to make this distinction from the point of view of prognosis and, ordinarily, simple methods are sufficient to indicate the proper differentiation. Even when the urine contains albumin and casts, if the specific gravity is high, the urine is of high color and there is no secondary anemia, it is extremely unlikely that the kidneys are involved to any important degree. Renal function studies may also be somewhat depressed as a result of passive congestion and return to normal when the cardiac condition improves. It will be helpful, therefore, to appraise the renal factor intelligently in order to make a proper prognosis when there is congestive heart failure.

There are other rarer forms of heart disease in which the prognosis may be excellent despite a considerable degree of heart failure, because treatment is so effective. This applies to cases of beri-beri heart, failure from arteriovenous fistula, constrictive pericarditis, and heart failure accompanying acute nephritis or toxemia of pregnancy.

Finally, in judging prognosis there are several types of complications that occur in heart disease which may be regarded as unexpected "accidents," which may suddenly change the outlook entirely. It often is



impossible to predict which patients will develop these complications or when they will occur. There are certain general principles, however, that enable us to divide our patients into those who are more likely and those who are less likely to have these unexpected "accidents." I now refer to the development of subacute bacterial endocarditis, emboli (pulmonary or arterial), the different forms of paroxysmal rapid heart action, heart block and coronary thrombosis. It is obvious that in a patient with heart disease who has been progressing most satisfactorily, the prognosis might suddenly become grave if one of these complications developed.

As to subacute bacterial endocarditis, one may expect that patients with well-marked mitral stenosis or hypertension as a group will only rarely develop it. If there has been persistent auricular fibrillation or a past history of congestive heart failure, it is extremely unlikely that the patient will ever have subacute bacterial endocarditis, no matter how long he lives. It also may be said that if there is no heart murmur, the patient not only has no subacute bacterial endocarditis but will not develop it in the immediate future. On the other hand, those most likely to succumb to this fatal disease are the patients who have been fairly strong, comparatively free from dyspnea or recurrent attacks of rheumatism, able to work and who have shown an apical systolic murmur of mitral insufficiency or a basal diastolic murmur of aortic insufficiency. In a word, it is the well-compensated patient with valvular disease, generally rheumatic, having the above murmurs and a regular heart, without hypertension who is most apt to develop subacute bacterial endocarditis.

When it comes to predicting which patients will have emboli the problem is even more difficult. In general it may be said that those with mitral stenosis are much more apt to have emboli either from the right auricle to the lungs or from the left auricle to the systemic circulation, than are those with aortic disease. The presence of auricular fibrillation with or without mitral stenosis is also conducive to the production of emboli from either auricle. Emboli from the ventricles (generally the left ventricle) occur almost exclusively following a coronary thrombosis and infarction of the ventricular musculature. I know of no method, however, of foretelling which ones of the above types of patients will actually have emboli. When such an "accident" occurs the prognosis may need to be suddenly changed.

The third type of "accident" in patients with heart disease is the sudden development of a new rhythm of the heart. A patient with complete heart block and a slow rate of 30 to 35 may carry on in good health for a great many years. Attacks of Adams-Stokes syncope, however, do occur unexpectedly and any one of the attacks may be fatal. It is impossible to predict when this may take place. A much more common "accident" in which the rhythm of the heart changes is paroxysmal rapid heart action. This may be due to auricular fibrillation, auricular flutter, auricular tachycardia or ventricular tachycardia. This is not the place to discuss the differential diagnosis of these arrhythmias except to state



that in most cases ordinary bedside methods suffice for their proper differentiation. When the heart suddenly becomes rapid the clinical condition often quickly changes for the worse, but the patient generally does better than the physician anticipates because many of these disturbances are transient and self-limited or respond most satisfactorily to treatment. Furthermore, there are many individuals who have no heart disease whatever, who suffer from these paroxysms of rapid heart action and carry on indefinitely with practically no restriction in activities. The important point with regard to these paroxysms is that the outlook is not as grave as it seems, for most of them respond to proper treatment.

Finally, we must consider angina pectoris and the "accident" of coronary thrombosis. There is no other condition in the practice of medicine in which it is so difficult to prognosticate. Patients who suffer from angina pectoris may die suddenly and unexpectedly shortly after they are first seen or may live for over twenty years. Such sudden fatalities may but need not be the result of an acute coronary thrombosis. In some the vessels, though showing atheroma, will not be thrombosed. It is even believed that sudden death may occur in angina pectoris with a normal heart and normal coronary arteries. If this does occur, it must be rare. Patients with angina are also prone to attacks of coronary thrombosis that do not cause sudden death. I know of no way of predicting when these accidents will occur. Furthermore, when a patient has an attack of coronary thrombosis there is no satisfactory method of foretelling which individual patient will survive and which will succumb. I have seen patients who were most seriously ill recover and others, who apparently were doing extremely well, suddenly die. The age at which other members of the family died of coronary disease at times is helpful in prognosis. Here the physician should remain hopeful under the darkest circumstances and yet give a guarded prognosis when the progress seems most favorable.

The foregoing are some of the general principles that I believe will prove valuable in formulating the prognosis in individual patients suffering from cardiac disorders. It is clear that much remains to be known but I have found that these generalizations have been extremely helpful and that their use will at least diminish the number of errors in prognosis, which errors are constantly placing the medical profession in disrepute in the minds of the general public and encouraging the work of the irregular pseudo-medical cults that pervade our country.



## THE NATURE AND TREATMENT OF CONGESTIVE HEART FAILURE

IT is no simple matter to analyze in detail the nature and exact mechanism of congestive heart failure. Despite exhaustive clinical, physiologic and pathologic studies there are many questions that remain unanswered. It may be of some value, however, to review some of the more generally accepted concepts that bear on this subject.

**Peripheral Circulatory Failure.**—At the outset the physician must clearly understand what conditions are not heart failure. The fact that the heart becomes rapid and the blood pressure falls does not necessarily mean that the heart is failing or that there is any heart disease. Patients who die of infections such as pneumonia, puerperal sepsis, typhoid fever and the like, die when the heart stops but death is not due to heart failure. The enfeebled state of the circulation is due to peripheral circulatory failure. Measures ordinarily employed to help the heart are generally useless and at times even harmful. In this condition the circulating blood volume diminishes; there is an inadequate return of blood from the periphery due to stagnation or pooling of blood in the capillary bed and venules. The heart is not apt to dilate and unless there are pulmonary complications the patient has no dyspnea, orthopnea or significant tissue edema.

Peripheral circulatory failure or shock is common in infectious diseases and is often the presenting problem following hemorrhage, severe trauma, burns, surgical operations and anesthesia, and in stuporous states such as diabetic coma and cerebral hemorrhage. Generally, patients with peripheral failure will show a cold, moist skin with blotchy cyanosis, rapid pulse and an anxious facial expression. The blood pressure often is low but the systolic level may at times be fairly well maintained while the diastolic level falls. The cause of this state is not altogether clear but that the primary difficulty in the circulation lies in the capillaries and the small peripheral vessels seems likely. Studies have shown that the minute output of blood from the heart in this condition is apt to be lowered, and that the venous pressure is decreased rather than increased.

As for treatment, digitalis is for the most part useless and probably harmful. It will not slow the heart rate under these circumstances and there is reason to believe that it will actually further diminish an already decreased cardiac output. In fact, none of the drugs we have are of great value. It would be desirable to increase the blood volume. For this purpose injections of 1000 c.c. or more of 5 per cent glucose in normal salt solution may be given subcutaneously or intravenously. Sometimes



transfusions of blood are indicated. The most effective method at present is the intravenous injection of plasma or human albumin. For acute emergencies caffeine sodium benzoate (0.5 gram or  $7\frac{1}{2}$  grains) or coramine (1 to 5 c.c.) administered intramuscularly or intravenously, may be employed. There may be rare occasions of acute circulatory collapse in which such heroic intravenous medication will be helpful and possibly life saving. There is hope that some of the newer preparations will be more effective in combatting the low blood pressure of circulatory collapse. For no matter what the cause, a maintained low blood pressure is dangerous in that it sets up a vicious circle from which recovery is extremely difficult if at all possible. Oliguria, nitrogenous retention and increasing stupor quickly result. There is some evidence that preparations like paredrine (10 to 20 milligrams given subcutaneously) will favorably counteract this state and produce an elevation in blood pressure without an increase in the work of the heart.

### THE NATURE OF CONGESTIVE HEART FAILURE

**Symptoms and Signs of Congestive Failure.**—It is only necessary to mention briefly the clinical evidence of congestive failure, as it is fairly well understood by most physicians. One should distinguish failure of one ventricle from that of the other, although both are frequently involved. Furthermore, it must be appreciated that subjective complaints generally precede physical signs as evidence of early heart failure. Patients may show murmurs, arrhythmias and cardiac enlargement for many years and remain well compensated. There are very few physical findings which of themselves would make one infer that heart failure is present or impending in the absence of symptoms. Amongst these are chiefly Cheyne-Stokes breathing, a diastolic gallop rhythm and possibly pulsus alternans, which may occasionally be elicited. Most of the other physical findings follow subjective complaints.

The earliest evidence of heart failure is abnormal breathlessness. The patient notices shortness of breath on effort that formerly produced no distress. This is due to failure of the left ventricle. In some, dyspnea may come suddenly at rest, particularly at night. The mechanism of these disturbances will be discussed. With increasing dyspnea the amount of effort that can be tolerated comfortably decreases, finally reaching the point of orthopnea or difficulty in lying recumbent. Cheyne-Stokes breathing is common. In the earliest stages physical examination of the lungs may reveal no abnormality, although the vital capacity of the lungs will already be diminished and x-ray may show some pulmonary congestion. Later, increase in the pulmonary second sound, basal râles and finally hydrothorax develop. In some cases a slight unproductive cough coming at night or on effort occurs as a result of pulmonary congestion.

The first evidence of right-sided failure is dependent edema. The ankles begin to swell as the day progresses, the swelling disappearing



overnight. Later, edema of the legs persists and extends upwards involving the thighs, buttocks, abdomen and occasionally the upper extremities and face. When there is considerable edema, it may involve the upper part of the body if the patient can lie flat, because of the effect of gravity. As a part of the venous congestion due to right heart failure, the venous pressure increases, the jugular veins become distended and the liver enlarges. Ascites may be present as a part of the general process of edema and partly as a result of portal obstruction. Due to the congestion of the liver there is often epigastric pain and other abdominal symptoms. In fact, occasionally pain in the right upper abdomen on walking may be an early or primary complaint, especially in patients with mitral stenosis, even in the absence of dyspnea or noticeable swelling of the legs. In such cases the degree of activity is not great enough to elicit breathlessness, while the increase in right-sided failure is sufficient to cause pain in the liver.

There are other general complaints that accompany heart failure but they are less distinctive and are met with too commonly in other conditions not associated with heart failure. Weakness, easy fatigability, insomnia, irritability, indigestion, palpitation and dull ache over the precordium have been emphasized by some authors as early evidence of weakening of the circulation. Although these are complaints frequently found in cardiacs, they rarely help in establishing the diagnosis of heart failure and in fact are often misinterpreted when due to other causes.

The combination of both right- and left-sided failure is seen more often than either one in its pure form. Obviously all degrees of heart failure will be found. In the advanced stage, cardiacs become cachectic showing marked loss of flesh, and cyanosis of the skin, especially of the distal portions of the body such as the ears, nose, cheeks, hands and feet. With this cyanosis there is often a slight icteric tint to the skin.

**The Mechanism of Congestive Failure.**—For generations there have been two diametrically opposed schools of thought concerning the mechanism of heart failure. According to one it is believed that heart failure is due to insufficient output of the heart to the tissues (forward failure) and to the other that it is due to congestion of parts of the body or back-pressure (backward failure). The arguments pro and con will not be discussed here, but it may serve a useful purpose to mention some of the factors in circulatory dynamics that bear on this question. They will aid in understanding the events we witness in practice and in rationalizing the therapy we employ.

One must distinguish some of the symptoms we often see in cardiac patients that may be looked upon as by-products, complications or accidents of the condition and not as fundamentally related to the question of heart failure. A patient may have attacks of Adams-Stokes syncope and yet have no evidence whatever of congestive failure, not even experiencing anginal pain or dyspnea on the most strenuous effort. Many cardiacs suffer from weakness. This may be due to many causes amongst



which a diminished cardiac output is important. But such weakness can occur without any congestive failure. This is seen during prolonged paroxysmal tachycardia, in which, because of an extremely rapid heart rate, the minute output of blood is markedly diminished. The patient feels quite weak but he may have neither pulmonary nor peripheral congestion. A similar situation in which there is marked weakness without congestive failure is seen in some cases of acute coronary thrombosis.

It is of primary importance to look upon the left and right portions of the heart separately for, although we generally see the results of failure of both sides, it is often possible to distinguish the phenomena characterizing the failure of each side separately. At times one will observe evidence of pure one-sided failure. The importance of a balance between the two ventricles becomes clear from the following analysis. If, as a result of any cause, the left ventricle expelled one drop of blood less per beat than the right ventricle, within three to four hours about one liter of additional blood would accumulate in the lungs. This would lead to acute pulmonary edema. A condition similar to this occurs in acute left ventricular failure with paroxysmal cardiac dyspnea. On the other hand, if the right ventricle expelled one drop less than the left an equal amount of blood would accumulate on the venous or right side of the circulation, resulting in venous distention, enlarged liver and, possibly, peripheral edema. However, if both ventricles diminished their output equally, although the circulation would slow up there would be no accumulation on either side and neither pulmonary congestion nor peripheral edema would result. In other words, although a slight imbalance of the two ventricles may exist for short periods of time, the two ventricles must eventually expel equal amounts of blood if disaster is to be avoided.

The processes involved in the gradual development of congestive heart failure are both numerous and intricate. Let us trace some of the events that occur during the progress of a case of mitral stenosis. As the mitral valve becomes increasingly narrowed the pressure in the left auricle increases. The wall of the left auricle becomes slightly dilated, which has two effects. It tends to make the pressure within its cavity return to normal and, following Starling's "law of the heart," the slight dilatation improves the contraction and compensates for the obstruction of the valve. The circulation is restored to normal but with the result that the left auricle is slightly larger, and eventually the pressure in the auricle and the pulmonary circuit becomes increased. As years go on and the condition progresses the right ventricle feels the burden of the increased pulmonary pressure. The pulmonary second sound increases in intensity. The right ventricle first dilates, thereby increasing its ability to expel blood, and later hypertrophies. It is a matter of general experience that prolonged dilatation precedes and is the stimulus for hypertrophy. In this way the bilateral balance of the two sides of the heart is maintained. The equilibrium that now exists in this patient with mitral stenosis is such that, although there is no peripheral congestive failure,



there has been a slight increase in the volume of blood in the lungs owing to the prolonged backward pressure behind the stenosed valve. At this stage the patient may be short of breath on effort but shows no râles in the lungs. The vital capacity of the lungs, however, may already have decreased. This measurement often is the first evidence of a diminished cardiac reserve and is the result of a decrease in the total available air space in the lungs.

In following the developments of the above case the progression may take different courses. If the right ventricle has become sufficiently hypertrophied and has not dilated too much, and the mitral obstruction is considerable, increasing breathlessness may result from further engorgement of the pulmonary circuit. In fact, such patients often have attacks of pulmonary edema. At such times, as a result of additional effort or of some nervous mechanism, or for some other ill-defined reason, the right ventricle pumps out more blood than the left and the difference is trapped in the lungs. An adjustment of the circulation occurs as the right side decreases or the left side increases its output. From this one can see how phlebotomy may occasionally be helpful in producing this adjustment.

The course of events, however, may be different. The right ventricle may dilate beyond the point of mechanical efficiency. With this there is an increase in the pressure in the right auricle, the venae cavae and the systemic veins. This increase in the venous pressure (normal values are 40 to 80 mm. of water) is the first evidence of right-sided failure. One of the earliest results of this is engorgement of the liver. Another is the development of pitting edema of the recumbent portions of the body. In some cases, despite the increase in venous pressure, peripheral pitting edema does not occur or is delayed because there are other factors concerned with the production of edema. Amongst these, one of the most important is the pumping action of muscular contraction of the legs on the venous return. If there has not been enough dyspnea to prevent the patient from walking, the exercise of the legs tends to delay the development of edema. Changes in the walls of the capillaries, the osmotic pressure of the blood, the development of acidosis, and to a lesser extent the question of tissue anoxemia, which is determined by the volume output of blood, all may have a bearing on the appearance of peripheral edema.

The above hypothetical case could therefore present itself at one stage as a patient with mitral stenosis complaining of dyspnea on exertion without physical signs of congestion but having a diminished vital capacity of the lungs. At another time this patient might have a good deal of obvious pulmonary congestion with marked breathlessness or he might show considerable venous distention, enlarged liver, with or without pitting edema, and little dyspnea. Finally, all the evidence of failure of both sides of the circulation may be present.

If an hypothetical case had started with hypertension or aortic valvular disease the primary strain would have been on the left ventricle.



At first, because of the increased work, the left ventricle would dilate a bit, and thereby compensate to eject the normal amount of blood into the aorta. If it did not do so for any significant length of time, acute pulmonary edema would result. In the course of time the prolonged dilatation and subsequent hypertrophy of the heart progress beyond the point of improved efficiency and the left ventricle fails to propel as much blood as the right ventricle. The result, as in the case of mitral stenosis, is increased pulmonary pressure, congestion of the lungs and dyspnea. The right ventricle, laboring against a constant increase in pressure in the pulmonary artery, becomes hypertrophied. This accounts for the frequent enlargement of the right ventricle that is found in cases of left ventricular strain. Finally, when the right side is sufficiently embarrassed, peripheral edema, engorged liver and ascites may develop. Patients who originally had primary left ventricular strain may be seen with various combinations of cardiac failure, some mainly with dyspnea, others with little or no dyspnea and showing for the most part peripheral congestion, and still others with different degrees of both.

There are other effects on the dynamics of the circulation and physiological functions of the various organs of the body that result from congestive heart failure. The total volume of blood increases. From a normal amount of about 5000 c.c. it may increase to six or seven liters or more. This is one of the most constant changes that accompany heart failure. This excess blood is stored throughout the venous side of the circulation, the lungs and to some extent in the dilated heart. The fact is often overlooked that when a heart is considerably dilated there may be as much as one liter or more of blood in excess of normal within its chambers. In such cases there must be a large amount of residual blood in the heart, for no increased amount is expelled with each systole. The total blood volume will always decrease and approach normal with clinical improvement. It has been difficult to explain what happens to the one or two liters of blood that disappears from the body as congestive failure improves. Recently, however, it has been shown that during congestion there is an increase in red blood cell formation as evidenced by reticulocytosis, and with improvement there is a diminution in blood formation and an increase in blood destruction. The latter is indicated by an increase in the icteric index of the blood and a marked increase in the urobilinogen in the stools. The increase in blood volume in congestive failure is so constant that occasionally it may serve as a diagnostic point. I recall an instance in which a man with definite organic valvular disease had a good deal of edema and pulmonary congestion. There was ample evidence that he had nephritis as well. At first the problem was regarded as one of heart failure. It was found, however, that the blood volume was perfectly normal. This and other studies of the dynamics of the circulation all showed that the patient could not have heart failure and that the edema must be due to nephritis. He died shortly thereafter of typical uremia.



Disturbances in the velocity of blood flow are of considerable importance in cardiac patients. In certain hyperdynamic states, such as obtain during fever, exercise, anemia and especially with hyperthyroidism, the rate of blood flow (not to be confused with the heart rate) is increased. Blood moves more rapidly from place to place in the circulation. With congestive failure the blood velocity is slowed up, mainly owing to the increase in the blood volume or circulatory bed. When slowing is considerable there is likely to be pulmonary congestion, but slight slowing can take place in constrictive pericarditis, when the lungs are not congested. One factor that has been neglected as causing a slowing in the velocity of blood flow as usually determined is the volume of the heart itself. There are instances of marked dilatation of the heart, especially of the auricles, when the cavities may contain two liters or more of blood. The circulation time will necessarily be markedly prolonged because of the dilution in this enormous residual pool and the slow movement of blood, even if there is no pulmonary or venous stasis. The methods in common use measure the time it takes for blood to flow from a vein at the elbow to the heart, through the lungs, out of the aorta to the tongue or respiratory center. Normally this requires about fifteen seconds. When delay occurs it takes place for the most part in the lungs or heart. Patients with marked congestion may show a velocity of blood flow (arm to tongue) of twenty-five to fifty seconds or more. In fact, if the rate of flow is normal it is extremely unlikely that there is any chronic cardiac congestion of the lungs.

There are various methods of measuring the velocity of blood flow that are in current use. Sodium dehydrocholate or decholin (3 to 5 c.c. of a 20 per cent solution) may be injected into an antecubital vein. The time from the injection to the arrival in the tongue is signaled by the sensation of a bitter taste. Similarly, 2 to 3 grams of saccharin in a few c.c. of sterile water may be injected. The end point in this case is a sweet taste. Sodium cyanide (0.25 to 0.5 c.c. of a 2 per cent aqueous solution) may also be used. The advantage of this method is that the end point is objective, for the patient will suddenly take deep gasping breaths the moment the drug reaches the carotid sinus. Alpha-lobeline hydrochloride (5 milligram—0.5 c.c. of 1 per cent solution) intravenously can be used. The end point is a sudden cough thought to come from carotid sinus stimulation. Calcium gluconate (2.5 c.c. of a 20 per cent solution) may be used, in which case the end point is a sensation of heat in the tongue or mouth. Caution must be exercised in injecting calcium when the patient has been receiving full doses of digitalis because of a synergistic action of the two drugs. In all these procedures the patient should be recumbent and relaxed with the arm lying at about the level of the heart. The injection should be rapid and the end point accurately timed with a stop watch.

The preceding methods measure the time it takes blood to flow from a vein in the arm through the lungs, back to the heart and out through the aorta. If it is desired to measure the time consumed in the flow of



blood through the lungs, the ether method is used. An injection of 0.5 to 0.5 c.c. of ether with an equal amount of normal saline is made into the brachial vein. The moment the patient or the observer detects the ether in the breath is the end point. This time, which normally is about four to eight seconds, subtracted from the arm to tongue time is an indirect measurement of the pulmonary circulation time because most of that delay takes place in the pulmonary vessels.

Measuring the velocity of blood flow is a simple procedure for differentiating many conditions such as emphysema, bronchitis, bronchopneumonia, cancer of the lungs, aneurysms, etc., from heart failure. Not infrequently the features produced by these conditions, such as dulness, râles, dyspnea and chest pain, are mistaken for congestive failure. In all of these states the velocity of blood flow is normal or not much changed, whereas if heart failure is present it is slowed. It is evident, therefore, that a prolonged circulation time through the lungs is an indirect method of detecting failure of the left ventricle. There is one important exception to this general rule. When *acute* pulmonary edema develops in a patient who previously was in fairly normal health, as may happen with acute coronary thrombosis or in a well-compensated hypertensive individual, the velocity of blood flow may be essentially normal. It apparently requires a more chronic state of passive congestion in the lungs, so that pulmonary blood volume is markedly increased, before slowing of the circulation may take place.

An increase in the venous pressure likewise affords an early measure of failure of the right ventricle. This can often be estimated by observing the degree of distention of the cervical veins. Normally these veins are completely collapsed with the patient in the upright or semirecumbent position. The height above the heart to which the distention rises serves as a rough measure of venous pressure. The exact reading can be made directly from the antecubital veins (normally about 40 to 80 mm. of water). In certain cases in which the question of congestive heart failure is in doubt, the determination of the venous pressure may be very helpful. Obviously an enlarged liver or ascites may be due to a malignant growth, alcoholic cirrhosis, inflammatory conditions or passive congestion. Similarly, edema of the legs may be due to nephritis, varicose veins or cardiac failure. In all such cases if the cardiac function is intact the venous pressure in the arms will be essentially normal.

One of the simplest and most valuable of the measurements that aid in estimating cardiac efficiency is the vital capacity of the lungs. Some of the factors that influence the vital capacity have already been discussed. (See Chapter 6.) All that is needed to obtain this reading is a spirometer. The apparatus is very simple, inexpensive and requires no upkeep. The test can be performed in one minute by any physician and will afford valuable information. It has been very much neglected and should be used by all practitioners. Once a diagnosis of heart disease has been made, the vital capacity determinations will be much more valuable in



estimating prognosis and in following the progress of a cardiac patient than the more expensive electrocardiograms that are frequently taken. Furthermore, the finding of a normal or supernormal vital capacity practically rules out congestive heart failure. This is particularly valuable in detecting cases of functional dyspnea when the readings are so often perfectly normal. Diminution in the breathing space is a very early sign of left ventricular failure. As has been stated, this may precede the appearance of basal râles. The decrease in the vital capacity results mainly from engorgement of the pulmonary vessels, which thereby diminishes the available alveolar spaces, and from a loss of elasticity of the bronchioles. Other factors in some cases are the development of hydrothorax and even the huge size of the heart. The volume of the heart occasionally may be actually one or two liters greater than normal. It is obvious under such circumstances that, with the thoracic cage remaining unchanged in size, the available space for ventilation must diminish considerably. Normally the average adult having a total breathing space of about 4000 c.c. inhales 400 to 500 c.c. with each breath and on effort can increase both the rate and the depth of respiration without distress. When the vital capacity is diminished to 2000 c.c. he reaches his greatest possible depth of inspiration more readily and will resort to a greater increase in rate of respiration to attain adequate ventilation. Furthermore, with these short and rapid breaths there is an increase in the proportion of the dead space (that upper part of the respiratory tract in which the air is not exposed to the blood for gaseous exchange). In this way a diminished vital capacity is conducive to dyspnea or uncomfortable breathing.

It is also of some interest that pulmonary congestion may set up certain vicious circles which tend to aggravate the very condition that produces them. Bouts of coughing, so common in cardiac failure, tend to increase still further the return flow of blood to the right side of the heart and thereby overburden the pulmonary circuit. The same is true of the act of overventilation or agitated breathing such as is seen during the period of hyperpnea of Cheyne-Stokes breathing. From this it is clear that whatever tends to quiet cough or vigorous breathing, *e.g.*, a hypodermic injection of morphine, not only makes the patient more comfortable but actually improves the underlying congestion.

There is a form of breathlessness common in certain cardiacs that is particularly prone to occur at night. It assumes forms of varying severity, from a slight increase in the degree of dyspnea to violent attacks of suffocation. It is often called "paroxysmal nocturnal dyspnea" or "cardiac asthma." It occurs most commonly in association with conditions in which there is a strain on the left ventricle, such as hypertension, aortic valvular disease and coronary artery disease. It is less frequent in cases of mitral stenosis. The factors that underlie this phenomenon are numerous. When the patient goes to bed there is a slight gradual shift of fluid from the periphery to the pulmonary circuit. Venous return from the legs becomes facilitated and the right ventricle expels a little more blood



than the weakened left ventricle can expel. There is also a slight increase in the metabolic rate of the body as the day progresses. This and the bodily activities during the day tend to produce greater pulmonary congestion. The vital capacity of the lungs which already had been diminished becomes further decreased. Furthermore, sensory perception is obviously diminished during sleep and the reflex stimuli from congested lungs, which are most important in the control of breathing, are less effective than during the waking hours. The result is that a greater degree of pulmonary stasis develops before the reflex stimuli from the lung are effective in producing exaggerated respirations. Finally the stimulus becomes sufficiently great to arouse the patient from sleep. The respiratory center now responds vigorously. The patient is then found to be breathing laboriously with evidence of slight or marked pulmonary edema. The cough that frequently accompanies the attack and the vigorous rapid breathing both tend to make the condition worse. The vicious circle may be broken by diminishing the return of venous blood to the right heart or by decreasing the sensitivity of the nervous system. The former is accomplished if the patient assumes the upright position or occasionally if phlebotomy is performed, and the latter by the use of morphine.

The foregoing discussion explains in general terms the mechanism of nocturnal dyspnea. There are, however, other less important influences involved which may serve as precipitating factors. The act of coughing, a full bladder, abdominal distention, unpleasant dreams, a change in environmental temperature, lowering the head, a large meal, etc., have all been observed to precipitate attacks of paroxysmal dyspnea in cardiac patients. By some method, probably through nervous reflexes, these factors serve as trigger mechanisms, but they all require a background of pulmonary congestion to be effective in the production of attacks of dyspnea.

Amongst patients who suffer from breathlessness, especially those with nocturnal dyspnea, and in some who are hardly aware that they have any dyspnea, periodic or Cheyne-Stokes breathing is quite common. Much attention has been given to its explanation but, although some light has been thrown on its mechanism, this peculiar phenomenon still remains obscure. It occurs mainly in patients with left ventricular failure, particularly in older people, and is most marked during, if not entirely confined to, the period of sleep. The breathing waxes and wanes with intervals of apnea varying in length from a few seconds to even a minute. The period of hyperpnea following the apnea may be very violent and awaken the patient in great agitation. In fact, this accounts for many attacks of nocturnal dyspnea.

Apart from the one predominant factor of pulmonary congestion there are other factors that have some bearing on the production of this type of periodic breathing. A diminution in the blood flow to the respiratory center, a decrease in the sensitivity of the respiratory center and of the



entire nervous system during sleep, an increase in intracranial pressure and a diminution in the carbon dioxide content of the blood may all play their respective roles. Once a tendency to periodicity develops it is not difficult to understand why it may continue or become more marked. During the apneic intervals the carbon dioxide level of the blood (which is the main stimulus for respiration) increases, as carbon dioxide is not being expired from the lungs. The oxygen content of the arterial blood is decreasing, as very little oxygen is being absorbed from the lungs. After a sufficient interval has elapsed the stimulus becomes great enough even to arouse the insensitive respiratory center, which responds in an exaggerated fashion. The result is violent hyperpnea which in time gets rid of the accumulated carbon dioxide from the blood through the lungs until the level is again too low to excite the respiratory center and the cycle begins again.

Morphine helps patients with Cheyne-Stokes breathing not by doing away with the periodic breathing, for it may even lengthen the periods of apnea, but by diminishing nervous sensitivity and thereby preventing patients from being aroused by the uncomfortable hyperpnea. Caffeine in large doses, on the other hand, may temporarily eliminate this type of breathing by increasing the sensitivity of the respiratory center. More recently, aminophyllin given intravenously in doses of 0.24 to 0.48 gram ( $3\frac{1}{2}$  to 7 grains) has been found very useful. What is more important are the measures that improve the underlying pulmonary congestion. When cardiac therapy succeeds in producing a diuresis, sometimes even one of slight degree, the condition may be greatly improved. Furthermore, the condition may be greatly helped by having the patient sit up in a chair with the legs hanging down rather than by keeping him in bed. This also tends to diminish the venous return to the right heart.

There are some other sequelae of congestive heart failure apart from those directly related to the dynamics of the circulation. Fever and leukocytosis are commonly seen in conjunction with cardiac congestion. In some instances an intercurrent infection may be the precipitating cause of the heart failure. Frequently, however, congestion itself produces slight fever. When the temperature is over  $101^{\circ}$  F. it is more likely to be due to some infection like bronchopneumonia, rheumatic fever or sore throat, or to infarction in some organ, especially the lung. The fact that slight fever ( $100^{\circ}$  to  $101^{\circ}$  F.) may result from uncomplicated heart failure, though commonly observed in ordinary cardiacs, is best illustrated in rare cases of paroxysmal tachycardia. Here one may see a patient without organic heart disease suddenly develop a rapid heart rate, and if the attack is prolonged he may gradually show evidence of pulmonary congestion and with it fever and leukocytosis. With disappearance of the congestion the temperature and white blood count return to normal.

Jaundice is another finding in some cases of heart failure. There are three factors involved in the production of this type of jaundice. In the



first place, passive congestion of the liver and anoxemia may impair liver function and disturb the excretion of bile through the bile ducts. Secondly, pulmonary infarction is frequently associated with jaundice through the breakdown of red blood cells in the lung. Finally, as congestion improves there is an increase in blood destruction. The icteric index in some cases may reach levels as high as 20 to 30 or more. As a result of prolonged hepatic congestion, cardiac cirrhosis may also develop. This may produce the added picture of portal obstruction to the already existing cardiac edema. In such cases repeated abdominal paracentesis may be required. Ascites may be quite conspicuous and out of proportion to the other evidences of heart failure. This occurs most frequently in cases of organic tricuspid stenosis and in constrictive pericarditis.

In determining the presence or absence of congestive heart failure, it is helpful to distinguish the objective from the subjective manifestations. Symptoms are more often misleading than signs. Breathlessness may be functional or bronchial in origin. Pain in the chest may be due to spondylitis and not to heart disease. Likewise, the objective signs may also have other causes than the heart. Peripheral edema may be due to varicose veins with its accompanying lymph stasis, râles to pneumonia, and enlarged liver to alcoholic cirrhosis or cancer. It is often much easier to ascertain whether heart disease is present than whether there is heart failure.

In observing and guiding the course of a patient with organic heart disease it is most important to bear in mind the factors that tend to precipitate heart failure. Inasmuch as the underlying original causes, such as rheumatic fever, arteriosclerosis and hypertension, cannot as yet be prevented, care must be exercised in protecting cardiacs against the aggravating or precipitating causes. The most important of these is infection. The others are for the most part those situations that unduly increase the work of the heart. Excessive physical effort, prolonged emotional strain, obesity, pregnancy, anemia, hyperthyroidism or any condition that accelerates the heart may be contributing factors. In so far as they can be prevented or remedied, just so far will ultimate heart failure be delayed.

The distinction has already been drawn between congestive heart failure and peripheral circulatory failure. It must also be made clear that both conditions may exist simultaneously. This is particularly true in some cases of acute coronary thrombosis and when organic cardiacs have intercurrent conditions such as pneumonia or cerebral hemorrhage. When both types of failure co-exist, difficulties arise concerning some of the methods of therapy commonly employed. A patient with severe acute coronary thrombosis may have pulmonary edema indicating left ventricular failure, and cold, gray, moist skin with a low blood pressure as a result of peripheral shock. For the former condition phlebotomy might be useful and for the latter one might reasonably give a transfusion. Likewise, digitalis is beneficial for the former and useless or harmful for



the latter. In such cases the physicians must weigh the advantages and disadvantages of the treatment in each individual instance.

**The Clinical Picture of Congestive Failure.**—A brief review of the clinical picture of congestive failure may now be summarized even at the risk of some repetition. The type and severity of complaints and the variety of physical findings will depend on the particular case involved and the stage of the process that has been reached. Symptoms as a rule precede signs. Breathlessness is the most important and generally the earliest evidence of heart failure. As has been discussed previously, it is necessary to rule out other causes, such as those of a functional and pulmonary nature, before regarding breathlessness as due entirely to the heart. In hypertension, aortic and coronary cases dyspnea may first appear at night, while in other cases it is first noted on effort. Cheyne-Stokes breathing especially during sleep almost always means heart failure. Even before dyspnea occurs most cardiacs complain of fatigue, "lack of pep," restlessness, insomnia and nervousness. These general complaints are too common in many other conditions, particularly in neurasthenia, to be very helpful diagnostically.

Edema of the ankles then develops. At first it is transient, disappearing overnight. Later it may persist. When orthopnea does not prevent the patient from assuming the recumbent position at night, edema may spread upwards earlier in the progress of the disease and involve the arms and face. The liver becomes engorged and causes pain or discomfort in the upper abdomen, and in the course of time ascites may develop. The amount of urine voided also decreases.

Examination will show various findings in different cases. Basal râles or diminution in respiratory excursion are amongst the earliest objective evidences of heart failure. Diminution in respiratory excursion is reflected in a diminution of the vital capacity of the lungs. The *x*-ray may show evidence of pulmonary congestion in the absence of râles. Later free fluid accumulates in the pleural cavities, especially the right. Auscultation of the heart may or may not reveal abnormalities depending on the nature of the lesion. Murmurs, irregularities and even cardiac enlargement, if present, are not necessarily indicative of heart failure, though they may signify the existence of heart disease. The presence of a diastolic gallop rhythm or pulsus alternans may generally be relied upon as signs of failure.

Cyanosis is apt to appear rather late. In some cases of mitral stenosis and in chronic cor pulmonale, it is often very marked. The cervical veins become distended and may remain so even when the patient is in the semirecumbent position. Gradually, when severe heart failure continues for years, a high degree of cardiac cachexia with marked wasting of tissue takes place. Such patients may show considerable swelling of the lower half of the body with very thin, emaciated arms and thorax. The urine, which is small in amount, may show albumin and casts, but unlike that seen in chronic nephritis it will be high colored and of a high specific



gravity. An unexpected gain of weight, while the food intake is actually small, often occurs and is due to the retention of fluid. This may take place without obvious edema, for cardiacs can have as much as five liters of excess fluid in the intercellular tissue spaces and yet show no pitting. Slight jaundice, often due to a complicating pulmonary infarction, is not uncommon.

Of greatest importance is the realization that most of the signs and symptoms of heart failure may be simulated in other non-cardiac conditions. An enlarged liver may be due to cirrhosis or cancer; pulmonary râles to pneumonia, bronchitis or tumor; edema of the legs to nephritis, pelvic tumor, varicose veins or hypoproteinemia; increased venous pressure to superior mediastinal obstruction, etc. One could further elaborate these similarities. It obviously is necessary to appraise the entire picture most carefully in determining the presence or absence of congestive heart failure.

**The Action of Digitalis.**—There is hardly a drug in medical use that has been studied so extensively as digitalis. Despite the most exhaustive pharmacological investigations concerning its mode of action, many questions remain unanswered. This discussion will be limited primarily to some of the known effects of digitalis and to the simpler aspects of its clinical indications.

Digitalis in sufficient doses increases the irritability of the heart as indicated by the production of ectopic ventricular beats, first few in number, then in the form of ventricular tachycardia and finally with the development of fatal ventricular fibrillation. In the experimental animal the latter mechanism is generally the cause of death when lethal doses are given. The therapeutic dose which averages about 35 to 40 per cent of the lethal dose causes none of these irregularities except possibly a few extrasystoles.

Digitalis also slows the conduction of impulses. This is especially noticeable in a prolongation of the P-R interval. The effect may be sufficient to result in partial heart block and rarely even in complete heart block. Occasionally it disturbs the conduction of impulses through the main or finer branches of the bundle of His, altering the form of the QRS waves.

Most of the slowing that results from digitalis and its allied drugs is produced by its stimulating effect on the vagus nerve. Apparently some of the slowing is extravagal for it is only partly abolished by full doses of atropine. In cases of auricular fibrillation, conspicuous slowing of ventricular rate results. This is brought about mainly by the action of the drug on the junctional conductive tissue. In hearts with a normal regular rhythm, only very slight slowing occurs. This slowing is so slight that it cannot be the cause of such striking improvement as is witnessed in many cases of heart failure with a normal rhythm.

There are other effects on the heart muscle produced by digitalis. Possibly the most important pharmacological effect of digitalis is that it



increases the strength or force of contraction of cardiac muscle. By direct action on heart muscle it lengthens the refractory period and slows the rate of propagation of impulses. These effects are counteracted by the indirect action of the drug through the vagus which shortens the refractory period and accelerates the transmission interval. The final effect is, therefore, variable, depending upon which influence predominates. The drug has another direct effect on the ventricular musculature that is shown in a peculiar inversion of the T wave of the electrocardiogram (see Chapter 21). It also causes a slight shortening of the Q-T interval. Animal experimentation has shown that, while therapeutic doses of digitalis produce no pathological changes in the heart muscle, toxic doses do cause definite necrosis of heart muscle fibers and inflammatory changes and also changes in brain cells. Whether these findings are in any way related to the previously mentioned electrocardiographic evidence of myocardial effects is doubtful.

It has long been known that digitalis diminishes the size of the heart. This has been ascribed to an increase in "tonus" of the heart. The possibility that this effect may be due to a peripheral mechanism was suggested by Dock and his co-workers. They showed that digitalis produces constriction of the hepatic veins. In this way it may be supposed that the drug pools blood in the liver and thereby diminishes the venous pressure and venous return to the right side of the heart, decreases the circulating blood volume and decreases heart size. In any case, one of the most constant actions of digitalis from a practical point of view is that of decreasing the size of the heart.

There has been much discussion concerning the action of digitalis on the coronary arteries and coronary blood flow. Most of the evidence points to a constricting action on these vessels due to a vagal effect. It is unlikely, however, that this results in diminished blood flow through the coronary arteries because compensatory effects may result from the accompanying slowing and other actions of the drug.

It is thought that digitalis improves the efficiency of the heart. By this is meant that it enables the heart to do the same work with less expenditure of energy. The evidence for this view is indirect and incomplete. More knowledge concerning the intimate metabolism of heart muscle is needed. It has been found that with heart failure the potassium and creatine content of heart muscle is diminished. It is apparent that, when the heart is unduly dilated, a decrease in size brought about by digitalis might well increase the mechanical efficiency of contraction. It can readily be seen that the effect of all these factors on the output of the heart will be variable. The decrease in venous return would tend to diminish and the improvement in cardiac efficiency or capacity would tend to increase the cardiac output. The former effect will predominate in cases in which the heart is normal or essentially normal and there is no abnormal residual blood in the heart, for no matter how efficient the heart may be it



cannot pump out more blood than it receives. When there is severe heart failure, however, the decrease in venous return is not a handicap and the improvement of the efficiency of contraction may be sufficient to produce an actual increase in the cardiac output. Direct measurements, in fact, have shown that digitalis actually diminishes the output of the heart in normal individuals and in patients with conditions like pneumonia where the heart is regarded as normal. In congestive failure, when the output may be either normal or somewhat diminished, digitalis has been found to produce a variable effect. In many cases there is an increase but in some a decrease in the output depending upon which of the two opposite effects predominates.

The main indication for the use of digitalis is congestive heart failure, whether it be left or right ventricular, or both. It does not matter then whether the blood pressure is high or low, whether the heart rate is rapid or slow, or whether the aortic or mitral valve is involved. When there is also a significant pulse deficit, as occurs in cases of auricular fibrillation or auricular flutter, apart from the previously mentioned effects, the drug improves the circulation by eliminating or diminishing the pulse deficit. When there is considerable cardiac hypertrophy, slowing of the rate is more important because a thick musculature needs a longer diastolic rest period for oxygen diffusion. It is open to question whether or not it is desirable to give digitalis continuously to those patients who have no heart failure but who manifest conditions that might lead to failure, such as hypertension with cardiac hypertrophy. If the circulatory dynamics are already normal and the patient has no symptoms it is likely that digitalis in therapeutic doses will upset the patient, and if there is no dilatation of the heart the cardiac output would probably be diminished by the drug rather than increased.

Digitalis is also used for long periods of time in those patients who recover from heart failure, to maintain the improvement that has been obtained, whenever the underlying condition is chronic and it is feared that heart failure might recur. There are disturbances in cardiac rhythm, not necessarily associated with congestive failure, for which digitalis may be used. Paroxysmal or maintained auricular fibrillation or auricular flutter and instances of paroxysmal auricular tachycardia may often be helped considerably by the judicious use of the drug. The rapid ventricular rate of auricular fibrillation or flutter may be slowed even when there is no heart failure, and especially in the latter condition a normal rhythm is often resumed. When attacks of paroxysmal auricular tachycardia recur frequently, constant administration of digitalis may prevent attacks entirely.

It is important to emphasize that digitalis not only is not indicated but may be harmful in cases of peripheral circulatory failure. In acute infections, surgical shock, heart tamponade from pericardial effusions or constrictive pericarditis, further diminution of cardiac output may result



from the drug. There are occasional instances in which the foregoing conditions are associated with true congestive failure and then digitalis may favorably affect the latter to a sufficient degree to be beneficial.

### THE TREATMENT OF CONGESTIVE HEART FAILURE

Unlike acute infectious diseases or many surgical conditions in which a complete cure is to be expected, the treatment of congestive heart failure is concerned with the amelioration of symptoms. Its purpose is to diminish suffering, to prolong life and to increase the usefulness of the patient for as long as possible. Although cures are not to be hoped for, because the underlying structural changes in the heart are for the most part irremediable, proper treatment may render individuals more comfortable, may restore some to useful occupation and occasionally may secure even real complete symptomatic recovery. For the most part the best that is accomplished by intelligent care of a patient with chronic cardiac disease is a prolongation of life that compares very favorably with the advantages that are derived from the early diagnosis and treatment of cancer. It is, therefore, with such restricted hopes that we must view this problem.

At the outset, the physician must bear in mind that social and economic factors often enter into consideration in outlining a course of treatment. This is as true of chronic heart disease as it is of many other chronic diseases. A day laborer who suffers from recurrent asthma and hay fever cannot be sent on a sea voyage or to the mountains every so often to rid himself of the offending agents and to avoid exposure to them. On the other hand, patients in more fortunate economic positions often derive considerable benefit from such expensive trips. Similarly those patients with angina pectoris, who are in a position to afford it, can go south in the winter and avoid the burden of cold winters and inclement weather. Likewise, the decision whether four or six weeks will be spent in the medical care of an individual case of cardiac failure may depend upon whether the man's job is jeopardized or whether he has sickness insurance, and other purely non-medical considerations.

Let us start with an average male of moderate means, forty years of age, whose occupation is that of bank teller. He has had mitral stenosis for many years and now presents himself in moderate congestive heart failure. There has been increasing dyspnea, some cough and, finally, peripheral edema. Physical examination shows moderate pitting of the ankles, a slightly engorged and tender liver, râles at the bases of the lungs and evidence of a slight right hydrothorax. The heart is dilated, the action is absolutely irregular, typical of auricular fibrillation, with a rate of 130 and the murmurs indicate the presence of mitral stenosis. The blood pressure is 150 mm. systolic and 90 mm. diastolic. The Wassermann is negative and the other findings are not significant. We may also assume that this patient previously had been receiving inadequate treatment and, in fact, had taken no digitalis whatever.



**Rest in Bed.**—The first principle in the treatment of such a patient is rest. Some patients, especially men, will rebel at what appears to them as such an extreme measure. They will plead to be permitted to cut down their work, to go into town for only one half a day or to remain at home resting or "taking it easy." It is best at the outset to explain that more will be accomplished in a shorter time if a strict regimen is carried out. An effective argument is to emphasize that they will start feeling better more quickly and will lose less time from work if they give themselves every advantage for recovery than if they go at it halfheartedly. I have found it helpful to explain that with complete rest, as compared with being merely ambulatory, the average heart saves about 25,000 beats each day and it is this enforced rest to the heart which is so beneficial. The advice "to go to bed" often carries with it a grave outlook in the mind of the patient. But the patient will have a better understanding of its significance and fear it less after a few days when he becomes aware of a distinct improvement. It is desirable to obtain as much mental and physical rest as possible and for this at times it is wise to delay starting the entire course of treatment for a day or two so that the patient may take care of certain matters that would otherwise prey on his mind.

When he takes to bed, simple devices should be used to make his bodily position comfortable. The proper number of pillows or a back rest, supports under the forearms and beneath the knees will aid considerably and often determine whether a patient will feel relaxed or restless. It may often be advisable to have him sit in a chair several hours daily with the feet hanging down, to prevent the shift of fluid from the periphery to the lungs. The use of a commode is often less of a burden than using a bed pan. During the early days visitors should be restricted, though diversions such as reading the newspapers and listening to the radio may be permitted.

I have become so convinced of the importance of the change in the dynamics of the circulation on assuming the recumbent position, which results in an increase in blood volume and an aggravation of the pulmonary congestion, that I now advise many patients to place wooden blocks under the head posts of their beds. These should be about 8 to 9 inches high and can be used indefinitely. This procedure is not particularly indicated when the main difficulty is hepatic congestion and edema of the legs or when the element of breathlessness is inconsequential. In some cases, moreover, it is better to have the patient stay in a chair all day.

**Diet.**—There are some differences of opinion concerning the proper dietary management of patients with congestive heart failure. I have found beneficial the simple Karel diet consisting of 200 c.c. of milk four times a day with no other food, but allowing a little more water or cracked ice for thirst. This is particularly worthwhile if the patient is overweight and has hypertension. This diet is easily taken and is low in



calories, salt, protein and fluid content. It often produces a feeling of restfulness and relaxation which improves the general condition even without any other medication. An inanition diet like this may have beneficial effects because of the same factors that are at work in normal people living on a low caloric intake. It has been found that semistarvation produces a fall in blood pressure, in pulse rate and in the basal metabolic rate. This no doubt may diminish the work of the heart and may thereby improve the circulation when failure is present. The milk diet should be continued for two or three days or even longer. After this brief period a more general diet containing the proper amount of vitamins is gradually allowed, restricting the salt intake and limiting the fluids to about 1500 c.c. A liberal intake of protein is permissible unless there is real evidence of nephritis. The total caloric intake will depend on whether it is planned to produce a loss of weight. Often this is desirable.

Some physicians are now advising an acid-ash diet for patients with congestive heart failure, restricting the sodium intake to an absolute minimum and permitting a liberal intake of fluids up to as much as 3000 c.c. After a very limited experience with this diet I am ready to heartily endorse it, for on occasions, what appeared to be dramatic results have occurred when other methods had failed. We have learned, at least, that much larger amounts of fluid may be given cardiacs with edema than formerly was the practice, and that the important restriction should be in sodium intake. In this regard cardiacs should be cautioned against using sodium bicarbonate.

The dietary management of patients with advanced heart disease took on a new aspect because of the interest in thyroidectomy as a method of treatment, which had for its purpose a diminution in the basal metabolic rate of the body. Proger proposed to obtain the same result by semistarvation. It was, therefore, suggested that patients suffering from severe heart failure be kept on a diet containing adequate vitamins and protein but only 500 to 800 total calories. Such diets are continued for months and possibly indefinitely. Some beneficial responses have been obtained. Although this method of treatment has its obvious disadvantages, considering the progressive nature of congestive heart failure and its therapeutic difficulties, one cannot refrain from paying some regard to this form of therapy.

**Sedatives.**—The care of the patient at night is a most important part of the treatment. If he spends a restless and sleepless night, struggling for air, he will have a poor day following it. Contrariwise if he obtains a good night's sleep he is apt to show improvement the next day. He is likely to need some sedative at night and if there has been nocturnal dyspnea it will generally be necessary to use morphine subcutaneously. Although the mildest sedative that is effective should be used, too much time should not be spent experimenting with bromides, phenobarbital, etc., during these early days. The first night a subcutaneous dose of 0.01 to 0.015 gram of morphine ( $\frac{1}{6}$  to  $\frac{1}{4}$  grain) is generally advisable. The



patient will often tell you that this is the first good night's sleep he has had in weeks. The dose should be diminished so that after three or four days some sedative may be given by mouth and will be found effective. By this time the condition of the circulation would have improved and comfortable nights might be anticipated by the use of 10 to 15 grains of sodium bromide,  $1\frac{1}{2}$  grains of phenobarbital or 15 grains of chloral hydrate. There is one type of heart failure in which morphine may be harmful and even dangerous. This is advanced cor pulmonale from emphysema or disseminated pulmonary arterial disease. The margin of respiratory function is so slight that further depression by the narcotic may be disastrous. Occasionally a cardiac will be seen when for one reason or another digitalis is not given and compensation of the circulation will become reestablished, primarily as the result of the proper use of sedatives at night. The care of cardiac patients at night is so important that the intelligence displayed in this regard may be the deciding factor in the recovery of the circulation.

Psychoses are common with severe heart failure, especially in elderly patients and those who have hypertensive or coronary heart failure. The manifestations are most marked at night and often present a difficult therapeutic problem. The irrational and panicky states in some patients appear to be made worse by narcotics. If the underlying cardiac therapy proves successful and congestive failure improves or disappears, the mental state almost always clears. It may be necessary to try omitting all sedation for a while or to discontinue morphine and give large doses of paraldehyde or chloral to obtain some rest. One must be ready to change from one course of treatment to another with the hope that the heart itself will eventually improve sufficiently so that no sedation will be necessary. In some of these cases I am inclined to give intramuscular injections of vitamin B complex daily on the suspicion that avitaminosis, which is not rare in severe cardiacs, may be playing a role.

**Care of Bowels.**—Vigorous catharsis used to be employed as a means of ridding a patient of edema. This practice fortunately has been discarded to a large extent. I have rarely seen any good come of it. In fact, during the first days of the treatment outlined here, while a patient is receiving very little solid food it does not matter if there is no bowel movement for two or three days provided there is no abdominal distention or discomfort. Thereafter a bowel movement every day or two, either occurring naturally or as a result of a mild cathartic or enema, is all that is necessary. Purgation is too exhausting and alters the edematous state too little to warrant its use.

**Digitalis.**—Digitalis is the most important drug in the treatment of congestive heart failure. Although it was first introduced into medical practice over one hundred and fifty years ago and its action has been studied in human beings and in animals by a good many students, the intricacies of its effects have not altogether been disentangled. Its indications and limitations have also been matters of heated controversies.



Despite this, at the present time our knowledge concerning its proper use is gradually becoming more firmly established and greater accuracy now exists concerning what may or may not be expected from it. Not so long ago it was taught that digitalis was contraindicated in aortic valvular disease, in febrile conditions and in hypertension. As to the latter it was feared that the blood pressure would be further elevated by digitalis. That fear no longer exists. In point of fact, when hypertension is present with cardiac failure the blood pressure generally falls during digitalis administration if the congestion disappears and when it does not fall the outlook is not so favorable. The view that prevails among most authorities is that digitalis is of use in any condition associated with congestive heart failure. In acute coronary thrombosis there is some doubt as to its possible beneficial or harmful effects. It certainly is indicated in patients with dyspnea and with peripheral pitting edema of cardiac origin no matter what the circumstances may be. There still is some dispute whether it acts beneficially in the presence of a regular heart beat. Many of the English clinicians were of the opinion, and some still are, that its use is limited to auricular fibrillation. In America it is generally accepted that, although the most dramatic responses are seen when auricular fibrillation is present, it has a decidedly beneficial effect even when the rhythm of the heart is regular. A more detailed discussion of the action of digitalis was taken up at the beginning of this chapter.

In using digitalis at the present time the physician is confronted with a great many different names and types of preparations. Over thirty years ago pharmacological studies showed that many of the preparations that were customarily used were below standard potency. In fact, some were found to be almost inert. The present situation is quite different. Now they are practically all satisfactory and carefully standardized by the manufacturer. The physician should choose a preparation that is not expensive for it may need to be used for a long time. He should not be misled into believing a special pill or a tincture will not produce nausea. They all can if they are active. The matter of preference for pills or liquid preparations depends a good deal on local habits. In England the tincture is still used a great deal whereas in America a pill or tablet of powdered digitalis leaves is more popular. The latter in general is preferable because of the difficulty in controlling accurate dosage if the tincture is used. It does not matter if the exact amount of sodium bicarbonate or many other medicines that are given is not known accurately, but it is imperative to know just how much digitalis is administered to our patients. There still prevails a misconception that 1 drop of tincture of digitalis is equivalent to 1 minim. I had the opportunity of looking into this matter during the First World War when it was my duty to standardize the digitalis preparations that were used in the American Expeditionary Force in France. I found that the number of drops necessary to make 1 c.c. of tincture of digitalis varied from 30 to 60. These variations depended on the size of the dropper, the speed with which the drops fell



and the angle at which the dropper was held. It is apparent from this that when a patient receives 5 drops three times a day, as is still prescribed by some practitioners, he may be receiving very inadequate doses. For these reasons and because of the convenience of administration, a pill containing 0.1 gram or  $1\frac{1}{2}$  grains of digitalis leaves (one cat unit) has proved to be a most suitable preparation. Occasionally one may suspect that because the pill is very hard it might not be digested in the alimentary tract (intact pills have been found in the stools). Under such circumstances a tincture may be used. At times one wonders whether the condition of the patient is not so low but that a pill would fail to be disintegrated in or absorbed by the alimentary tract. Here also one may properly try a liquid preparation. Finally individual patients occasionally find that they have less local irritation of the stomach taking one type than another type of digitalis.

Opinions still prevail that certain digitalis preparations will be beneficial when others are not. Careful studies, however, have shown that the margin of safety, *i.e.*, the difference between the therapeutic and toxic or lethal dose, is essentially the same for preparations like digitalis, digitoxin, lanatoside-C, strophanthin or ouabain when given intravenously. Confusion has resulted because absorption of these preparations varies considerably when given orally. Ouabain, a pure crystalline glucoside, is absorbed very poorly from the gastro-intestinal tract but is suitable for intravenous use (0.25 milligram once or twice the first day and once daily thereafter). Similarly, lanatoside-C (*digitalis lanata*) is useful for parenteral use, the full digitalizing dose intravenously being 1.5 to 2.0 milligram. The oral dose, however, is much greater. Six to eight milligrams given in divided doses during two to three days will be adequate and the daily maintenance dose is 1.0 to 1.5 milligram (2 to 3 tablets of 0.5 milligram). On the other hand, a preparation like digitoxin (*Digitaline Nativelle*) is completely and readily absorbed by the gastro-intestinal tract, so that the digitalizing oral and intravenous dose is practically the same, *i.e.*, 1.25 milligrams. It is of some interest that this dose is only 3 cat units and is equal in potency to 15 cat units of the tincture or the whole leaf when given orally. The reason for this discrepancy is that it is all absorbed, whereas about four-fifths of the ordinary digitalis given by mouth is non-absorbable. When digitoxin is used the full therapeutic dose of 1.25 milligrams may be given orally in one dose and then continued with a daily maintenance dose of 0.1 to 0.2 milligram. It is now thought that nausea will be much less common with this preparation.

Let us now return to the treatment of the patient mentioned above and let us assume that he had not received any digitalis before. The average adult will require about 2 grams or 30 grains of digitalis or 20 c.c. of tincture of digitalis given over several days. (The dose required in infants and children is apt to be one and one-half to twice as great in proportion to weight as in an adult.) The exact amount will vary, some



patients requiring less and some more. This dose can be given quite rapidly, even within twenty-four hours, but it is very rarely advisable to do so for it may turn out that only 1.5 grams or less were necessary and the remainder of the dose would have proved excessive and would produce toxic manifestations. When the situation is very urgent and an effect is to be produced in hours rather than in days the intramuscular or intravenous route should be used instead of the oral. There is no single method of dosage that needs to be followed. Much will depend on the condition of the patient and the frequency with which he will be observed by the physician. Generally it is satisfactory to give one-fourth of the total dose or 0.5 gram the first day and a similar amount the second day. One pill (0.1 gram each) five times a day will accomplish this. On the third day the dose may need to be diminished. If the apex rate, which was 130, has fallen to 100 it may be planned to give 0.1 gram three times on this day and after two or three days this may be cut down to one pill a day. The hope is to lower the apex rate to 60 or 70 if possible, without producing any ill-effects. If this patient is to be seen only once a day this is best done in the morning, for then one can witness all the good or harm that the previous dosage has produced and one can outline the amount to be taken during the following twelve hours. Finally the so-called "maintenance dose" of 0.1 gram daily is kept up. This is approximately the amount that is utilized or eliminated daily.

The course outlined in the preceding paragraph needs to be modified under certain circumstances. If the patient had received digitalis before it is better to start with smaller amounts. One pill three times a day may then be sufficient. Or he may be complaining of nausea and vomiting and doubt arises whether he has already received too much digitalis. More often than not these symptoms are due to circulatory failure, generally accompanied by hepatic engorgement, and demand more rather than less digitalis. If auricular fibrillation is present and the heart rate is still rapid one can feel quite certain that more digitalis is needed. This same conclusion cannot be drawn if the rhythm is regular, for the rate then may remain rapid even after full doses of digitalis are administered. Occasionally the electrocardiograms may be helpful in deciding the question although they are rarely necessary. When nausea and vomiting are present and further digitalis is to be given it is best to use the tincture rectally. Three or 4 c.c. (diluted in 50 to 100 c.c. of water) may then be given daily. I have used this method with success and the vomiting has ceased even in patients who were convinced that it was produced by the previous digitalis that had been taken. The dosage for rectal administration is the same as that for oral.

When great speed of action is required some form of digitalis should be used intramuscularly or intravenously. Such preparations are put up in ampules and generally contain 0.1 gram for each 1 c.c. or 2 c.c. Whereas most physicians are familiar with the proper oral dosage, the



situation is quite different with regard to the hypodermic use of the drug. In fact the exact dose with the latter method is not so well known. If 2 grams given orally is a proper digitalizing dose for a patient it certainly would be excessive if it were all given intramuscularly or intravenously in one injection. On the other hand, 1 ampule containing 0.1 gram is often given hypodermically, especially by surgeons, hoping to give the patient the benefit of digitalis. It is obvious that this dose is practically valueless. The proper amount to obtain an appreciable therapeutic effect is between 0.5 and 1 gram given either intramuscularly or intravenously. An effect may be expected with the former route in one to two hours and with the latter in fifteen to thirty minutes. The same dangers exist, however, when digitalis is given intravenously as when strophanthin or ouabain is used, for the margin of safety between the minimal toxic and minimal lethal dose is the same for all these preparations. The main danger is that if the patient has recently been taking any appreciable amounts of digitalis, intravenous preparations should be given with great caution and in smaller doses.

The following experience illustrates the occasional instance when rapid digitalization is imperative. Some years ago I was called to see a woman about forty years of age who was in a moribund state. She had mitral stenosis with a regular rhythm and had been ambulatory and getting along very well. She had not been taking digitalis. That day at 9 p. m. she was suddenly stricken with palpitation and dyspnea and in a short time developed acute pulmonary edema. When I saw her at 11 p. m. she was unconscious. There was marked cyanosis, stertorous breathing and the lungs were full of moist râles. The pulse was imperceptible but the heart rate as counted at the apex was about 190 and the rhythm was absolutely irregular. She had already received strychnine, camphor and caffeine hypodermically. This was exactly the condition in which digitalis was indicated, but even minutes were precious. I, therefore, gave her 8 c.c. (0.8 gram) of digitalis intravenously and in about twenty minutes the heart rate fell to 100 although the rhythm remained absolutely irregular. The effect was most dramatic, the breathing quieted down and most of the râles disappeared. A short time later the patient returned to her customary duties and presented the picture of a patient with well-compensated rheumatic mitral stenosis and auricular fibrillation.

It is always important to watch for the indications to diminish or to omit digitalis. The first obvious reason for cutting down the dose is if the desired therapeutic effect is produced. In the case of the bank teller (cited above), if the heart rate slowed to 70, the dyspnea and edema disappeared and the patient was subjectively improved, the dose should be reduced to about 0.1 gram a day. One, therefore, does not need to push the dosage to obtain toxic effects if the therapeutic result is satisfactory. The second reason for omitting the drug is if evidence of intoxication is detected. This may be either subjective or objective. It is curious



how patients differ in their reactions to digitalis. Some will quickly develop the subjective symptoms without the customary objective signs of intoxication and others do just the reverse. There are still others who on very large doses develop neither.

The most common early evidence of digitalis intoxication is loss of appetite, nausea and vomiting. There is often a general mean and sickly feeling, accompanying the desire to vomit. When this occurs there is no treatment except omission of the drug. Other symptoms that are less common are diarrhea or yellow vision. The objective evidence of intoxication concerns the findings in the heart. Among these are undue slowing of the rate of the heart, *i.e.*, under 50, the development of extrasystoles, previously not present, especially in the form of coupled beats, and heart block. The most common finding is digitalis coupling. Here every second beat is a ventricular extrasystole. This can easily be recognized if the rhythm of the heart was regular at the start but can also be detected at the bedside if auricular fibrillation is present. Any of three forms of heart block may result, first degree (delayed conduction time), second degree (partial block) or complete block. The first cannot readily be detected without an electrocardiogram, the second is easily recognized by noting an occasional dropped beat on auscultation, and complete heart block brought on by digitalis offers real diagnostic difficulties. The ventricular rate of complete heart block produced by digitalis is not around 30, as it is in Adams-Stokes disease. It is generally 60 or more and may be over 80 and 100. This regular rate will also continue to accelerate on increasing doses of digitalis. In animal experiments this is one of the mechanisms by which death from digitalis intoxication occurs, although the more common mechanism is ventricular fibrillation. As a practical matter, the first two types of heart block from digitalis do no harm and, in so far as slowing of the ventricle results, may be beneficial. They serve merely as a signal that further dosage must be given cautiously. Complete block, although rare, is an indication that the drug should be omitted or the dose should be diminished considerably. Inasmuch as it may be difficult to detect this clinically it is well to omit digitalis if the heart, which was previously grossly irregular, becomes regular on digitalis, until the exact mechanism of the heart beat is known. In such a case, regularization may be either due to a resumption of the normal rhythm of the heart, or the ventricles may be beating regularly while the auricles are still fibrillating. In the first instance nothing would be lost by omitting digitalis for a few days and in the latter harm might result by continuing its use. Electrocardiograms might be necessary in order that some of these unusual decisions may be made. There are also electrocardiographic changes in the T wave of the electrocardiograms as a result of digitalis (Chapter 21, Fig. 133), which do not indicate intoxication, that occasionally may help in estimating the amount of the drug that a patient has received.

Apart from digitalis or its allied drugs, like urginin, there is little



else in the form of medication that acts directly on the heart, which one can use in the treatment of congestive failure. Strychnine, camphor and caffeine had a considerable vogue in the past but there is very little satisfactory evidence to support their use, except that caffeine may serve at times as a respiratory stimulus. Adrenalin and ephedrine are useful in those cases in which the dyspnea or cough is in part due to an asthmatic or emphysematous state. Potassium salts (such as potassium phosphate, 2 grams three times a day) have been recommended on the basis that there is a deficiency of potassium in the myocardium in cases of heart failure. Much cannot be expected from their use. Although many other drugs have supposed beneficial effects they prove to be hardly worth the trial.

**Diuretics.**—Next in importance to digitalis in the treatment of patients with congestive failure is the group of diuretic drugs. Some of these are given by mouth and others can only be administered hypodermically. Among the former are theophyllin (theocin) and theobromine sodium salicylate (diuretin). Diuretics are best given after complete digitalization has been accomplished, if there still remains evidence of peripheral edema. It must be remembered that patients may have considerable latent edema in the body after all obvious pitting has disappeared. Furthermore, when the subject has been in bed for some time, it is well to look for subcutaneous edema in the sacral region, for fluid may accumulate there when the ankles are entirely free of edema. Theophyllin may be given in doses of 0.2 to 0.3 gram three times a day for one day and repeated every five to seven days if desirable effects result. This may produce nausea and vomiting and when they are marked the drug should not be continued. If an effect is produced with these doses it occurs within the first twenty-four hours. Sometimes several liters of urine will be voided in one day. Following this there may be fatigue of the kidneys so that these large doses should not be repeated for about one week. In ambulatory patients with a tendency to recurrent edema, a single daily dose of 0.3 gram of theophyllin is often well tolerated, together with the maintenance dose of digitalis, and produces just enough diuretic effect to prevent or postpone the necessity of abdominal paracentesis or intramuscular diuretics. Theobromine sodium salicylate (diuretin) acts like theocin and also may produce distressing nausea. The dose of this is 1 to 2 grams three times a day, once a week, or 0.5 gram twice a day for a more moderate but continuous effect. On rare occasions I have obtained a most satisfactory diuresis by giving 0.3 gram theophyllin intravenously well diluted in 10 to 20 c.c. of water. This can produce nausea just as the drug does when given by mouth. Similarly, aminophyllin, either orally or hypodermically, has been used for diuretic purposes. The oral dose is 0.1 to 0.2 gram *t.i.d.* and the intramuscular or intravenous dose is 0.24 to 0.48 gram. The difficulty is that in rare instances of coronary thrombosis fatalities apparently have been precipitated several minutes after intravenous injection of aminophyllin. However, this



method of treatment is often effective in the relief or the prevention of paroxysmal cardiac dyspnea.

During the past decade or so mercury diuretics have come into use and to a large extent are superseding those just mentioned. Of these salyrgan, mercupurin and mercurhydrin are the most commonly employed. They are given in doses of 0.5 to 2.0 c.c. intramuscularly, intravenously or by rectum in the form of suppositories. If even a small amount of the drug leaks out of the vein or is given subcutaneously there is considerable pain and an ugly and stubborn ulcer may develop and last for many weeks. Great care, therefore, must be exercised in administration. There is less risk of any harmful effects if the diuretic is diluted with 10 c.c. of sterile salt solution when the intravenous route is used. If the patient has veins that are difficult to find or if there is any doubt whether the needle is actually in the vein, it is better to withdraw the needle and give the solution intramuscularly. I have had occasional experiences, however, in which it seemed that no diuresis resulted from intramuscular injections, possibly because the tissues were so edematous, when intravenous injections were very effective. For the general practitioner the preferable method is to inject the drug deep into the gluteal region. It should not be injected into the muscles of the arm. This also is true for other intramuscular preparations, such as caffeine and digitalis, for soreness may develop in the arm and not in the buttocks. The dose will vary and as often happens it may need to be repeated every week or so for a long time. Under these circumstances it may be necessary gradually to increase the amount. I have seen a patient who had about 250 weekly injections over a course of five years always obtaining a most satisfactory diuresis of 5000 to 8000 c.c. in twenty-four hours and during this time the individual dose was gradually increased from 1 c.c. to 4 c.c. At no time in this case was there any evidence of renal damage despite the enormous amounts of mercury that were used. The mercury suppositories that are available have been effective in some patients but in others they have caused distressing irritation of the rectum and had to be discontinued.

One would very much welcome a mercury preparation that would serve as an effective diuretic when given orally. There is one that is being tried called "S.T.O." which has proved of some value in an occasional case. One to three tablets are given three times daily. Each tablet contains 0.08 grams of salyrgan and 0.04 grams of theophyllin.

In some cases the diuretic effect of salyrgan and mercupurin may be enhanced by the preliminary administration of ammonium salts. Either ammonium chloride or ammonium nitrate in doses of 1 to 2 grams (15 to 30 grains) four times a day for about four days followed by the injection of 1 c.c. of the mercury diuretic may be more effective than the mercury diuretic without the preliminary ammonium salts. In fact, occasionally a diuresis may be produced as a result of the ammonium salts alone. The action is supposed to result from the acidifying effect on the body.



These large doses of ammonium preparation are rather disagreeable but the annoying taste may be avoided if enteric-coated tablets are given. Another procedure is to give 2.0 grams of ammonium chloride once or twice, two hours before each mercury injection. Ammonium chloride may be administered more or less indefinitely with the maintenance dose of digitalis in order to retard the tendency to recurrent edema.

Two other methods of producing diuresis may be employed. Two to 3 ounces (60 to 90 grams) of urea in a single daily dose over long periods of time can be used with beneficial effects. Because of the nauseating effect of urea this will not be tolerated by most patients but for those who are not upset, it has proved of some value. The disagreeable taste of urea may be somewhat disguised if it is dissolved in orange or grapefruit juice or in ginger ale. It has also been recommended that all the sodium chloride be omitted from the diet and be replaced by 5 grams (75 grains) of potassium chloride daily. I have occasionally found this to be quite useful though it may produce uncomfortable abdominal cramps.

In general the various diuretics have added considerably to the treatment of patients with congestive heart failure. When properly administered it may be expected that beneficial effects will result if the function of the kidneys is good. At times it is no simple matter to anticipate without elaborate functional tests whether the kidney function is adequate or not. Albumin and casts in the urine may result from passive congestion of otherwise fairly healthy kidneys as well as from true nephritis. In general practice, when it is not easy to perform chemical analysis of the blood, the absence of any anemia and the presence of a high specific gravity of the urine points to a satisfactory functional state of the kidneys even when albumin and casts are present. Only occasionally will it be necessary to determine the blood urea nitrogen or non-protein nitrogen, or to perform the phthalein test or a concentration test to ascertain more accurately the state of the kidneys. Even when it is found that the kidneys are slightly or moderately impaired, if there is congestive heart failure and edema persists despite digitalis and bed rest, diuretics may be used. Although the results will not be as beneficial as when the kidneys are normal, such patients may yet do better following the use of mercupurin than without its use.

Cardiac cachexia and inanition are not infrequent results of long-standing chronic heart failure and with this there may be an element of edema, purely as a result of the low protein content of the blood, just as occurs in nephrosis. In fact, on occasions, because of the presence of considerable edema and certain other findings, a patient may be regarded as having heart failure when the edema is entirely due to nephrosis, nephritis, hypoproteinemia or some deficiency disease. It is now well known that a fall in the total protein content of the blood serum may produce edema. This is particularly true if the albumin content is markedly diminished, resulting in a reversal in the albumin-globulin ratio. Such changes diminish the osmotic pressure in the blood and permit fluid to



move from the blood into the tissue spaces. Such hypoproteinemia may result from prolonged dietary deficiency, especially when the protein intake is restricted, from loss of albumin in urine and also from liver disease. These factors not infrequently are involved in chronic cardiacs. The diets of these patients are often unwisely restricted by physicians or the patients do not eat because of persistent anorexia, and the liver is frequently in an unhealthy state from passive congestion. The result is that some cardiacs have edema not only because of decompensation but because of hypoproteinemia. Examination of the blood must be made in all cases of stubborn edema to establish the correct diagnosis. In some cases the edema will not be controlled until special efforts, such as the institution of a high protein diet or infusions of blood or, preferably, plasma, are made. When intravenous injections are difficult or impossible, infusions of blood or albumin may be given intrasternally, an 18-gauge needle being used and the injection being given in the mid-line.

Occasionally one observes patients in whom adequate response is obtained from the use of mercury diuretics but who steadily lose ground. As they become edema-free their general condition grows worse. Some seem to grow apathetic, drowsy and weak. Although this complication has not been thoroughly studied it may be that when it occurs there probably is an increase in the renal insufficiency and dehydration of the tissues. If this is so, some of the harmful effects might be obviated by increasing the amount of fluid in the diet. When the kidneys are impaired in function and unable to concentrate well it requires a larger volume of fluid intake and of urine in order that the necessary waste products be excreted. In fact, the prevailing custom of marked restriction in the intake of fluids for patients with congestive heart failure may need slight revision because of this. One other possible explanation of the ill-effects that are occasionally seen following a brisk diuresis is that the fluids stored in the body may contain considerable amounts of digitalis and when this is absorbed into the blood stream for excretion through the kidneys the patient is really getting a new large dose of digitalis from within, *i.e.*, he is being redigitalized. Finally, disturbances in the salt metabolism may prove to account partly for some of the ill-effects following diuresis.

Fever is common with congestive failure and is an added burden on the heart. Whenever this might be due to an infection responsive to specific therapy, such as the sulfa drugs, appropriate measures must be carried out. Occasionally it will be advisable to use a non-specific antipyretic, such as amidopyrine (0.2 to 0.3 grams), for short periods of time to diminish the work of the heart.

**Mechanical Methods of Treatment.**—There are occasional instances of congestive heart failure in which it is observed that a patient, previously ambulatory and showing considerable edema of the legs but only slight breathlessness, after being put to bed loses the peripheral



edema but develops marked dyspnea. As the right-sided failure improved the left-sided failure grew worse. The recumbent position favored the return flow from the periphery, but improvement in the left ventricle was not sufficient to take care of the greater venous return from the right ventricle and so increased pulmonary congestion took place. The fluid left the legs but appeared in the pleural cavities and lungs. The reason that this does not happen more often as patients are put to bed is that the increased efficiency of the left ventricle keeps pace with the increased burden and a diuresis occurs. The practical inference from these observations is that in certain instances it is advisable not to keep the patient in bed but to allow him to sit in a chair with the legs hanging down. It is much better to have fluid in the limbs than in the chest. I have seen patients suffering from extreme breathlessness, despite oxygen therapy and all other customary methods of treatment, promptly improve and recover on getting out of bed into a chair. In such a case one will observe dyspnea disappear while peripheral edema reappears. The latter can be effectively treated later.

In cases of acute pulmonary congestion in which circumstances might indicate the need of phlebotomy, tourniquets may be applied to the four extremities. Pressure should be about 40 to 50 mm. of mercury, or enough to prevent return flow of venous blood and yet to permit free forward arterial flow. This traps the blood in the periphery and pressure may be maintained for a half hour to several hours using the following method. Tourniquets are applied on three of the four extremities and every fifteen minutes one is released and applied on the limb that had been free. In this way each extremity has a rest-period every forty-five minutes.

Edematous fluid in the limbs can at times be removed by mechanical means. Southey tubes may be inserted subcutaneously under aseptic precautions into the swollen feet or hands. In this way several hundred c.c. of fluid or more may be lost daily and the edema thereby diminished. I once saw a patient who lost over 15 liters of fluid in three days by this method.

There remain several other procedures that are helpful in the treatment of patients with congestive heart failure. It is not uncommon to forget that a diuresis may occur and the urine remain in the bladder. This is particularly to be watched for in elderly males. I recall the humiliation I experienced in finding 1500 c.c. of urine in the bladder when the postmortem examination was performed in a patient I had treated for heart failure. The retention of urine in the bladder results primarily from prostatic obstruction. Although patient and physician may have been aware of this prostatic difficulty before, at times the first significant evidence of it may occur during the bed treatment for cardiac failure. It is obvious that the temporary remedy for this is catheterization.

A more common complication is hydrothorax. Most patients with advanced heart failure have some free fluid in the pleural cavities. When



the amount is small, *i.e.*, 100 to 200 c.c., it is not detectable nor is it of any advantage to remove it mechanically. But when there is over 500 c.c., or especially 1000 c.c. or more, considerable respiratory and general relief can be obtained by a thoracentesis. The bases of the lungs, especially the right, should always be carefully examined in all cases. Dulness or flatness on percussion, diminished breath sounds and decreased tactile fremitus with or without râles are the findings that help in detecting free fluid at the bases of the lungs. Tapping should be performed if it is expected to obtain more than 500 c.c. of fluid. There is no advantage in withdrawing the very last amounts and the needle should be removed when the fluid begins to come with difficulty or when an uncomfortable cough is produced. It is always best to use a dull needle so that unnecessary scratching or bleeding of the pleurae will not take place.

Abdominal paracentesis should also be employed when there is significant ascites. Here also small amounts of fluid are better left undisturbed. Tapping is indicated if the amount that might be obtained is more than 2000 c.c. The removal of smaller amounts does not seem to improve the condition sufficiently to make the procedure worthwhile. Furthermore, small amounts either in the abdominal or pleural cavities can readily disappear in the course of the routine treatment for heart failure, whereas the mechanical removal of larger quantities may expedite recovery. When there is marked ascites, mercury diuretics may be ineffective until the abdominal fluid is removed by tapping. It would appear that the pressure of the fluid on the renal vessels prevented the mercury from producing its customary effect.

Oxygen therapy may help patients with congestive failure. This is of particular value for brief periods of time during cardiac emergencies when there is severe breathlessness and pulmonary congestion. It is obviously not a procedure that is applicable in most cases of chronic failure which continues for months. However, I observed a middle-aged woman, who had advanced irreversible congestive failure with striking cyanosis, dyspnea and a very large liver, carry on her work as a school teacher for a few years. Apart from the customary cardiac medication she spent about two hours each day inhaling oxygen through a mask. She was convinced that the oxygen enabled her to carry on.

Finally a word must be said about phlebotomy. As we all know, blood-letting is an old method of treatment that was in vogue for centuries and used in all sorts of diseases. Much of this practice has been discarded and I am of the opinion that at present its application in the treatment of congestive heart failure has not been sufficiently utilized. In many of these patients, if not in all, the total volume of blood is increased. The various organs, especially the lungs and the liver, are markedly engorged. The pressure in the venous side of the circulation is increased as is manifested by prominent distended veins in the neck and an increase in the venous pressure readings taken directly from peripheral veins.



There is both experimental and clinical evidence that after a phlebotomy the state of the circulation can be improved in some cases. Some observations made years ago showed that the removal of 500 c.c. of blood from one arm improved the flow of blood in the other arm. I have witnessed a decided decrease in the size of the liver and a prompt disappearance of pain and tenderness in that region directly after the removal of 700 c.c. of blood in a patient with mitral stenosis and auricular fibrillation. What is much more striking is the effect of phlebotomy in some moribund cases. The following experience illustrates very graphically some of the results that may occasionally be obtained by this neglected method of treatment. I was once called to see a woman about seventy-three years of age who had asthmatic bronchitis, hypertension and myocardial failure. All the customary treatment had been employed and when I arrived the patient was moribund, unconsciousness having gradually developed that day. In fact, the breathing was such as is witnessed in patients only a few minutes before they die. The chin dropped with each breath and there were tracheal rattles. There was marked cyanosis and pulmonary edema. The situation was so desperate that I did not even sterilize the needle that was used to puncture the vein. About 600 c.c. of blood were removed in twelve minutes and just as the procedure was completed the patient became conscious. In two weeks she was out of bed and the recovery enabled her to live in comparative comfort for another eighteen months. In a second instance of this sort an elderly man who had been in a coma immediately regained consciousness after a phlebotomy of 550 c.c. of blood. Here the children made the simple request that they wished to speak to their father once more. He remained mentally clear that day, then relapsed into a coma and died the following day. In these two and other similar cases the effect must have been a specific result of the bleeding because all other methods of treatment had failed and the improvement occurred literally minutes after the operation was started. The amount of blood to be removed may be gauged by the level of venous pressure. This should not be reduced below normal.

The exact indications for phlebotomy are not clearly defined. Beneficial results are not always obtainable even when the conditions appear to be similar. The following conditions seem to be those in which bleeding may be helpful: engorged or tender liver, distention of the veins of the neck, cyanosis and pulmonary edema. When the blood pressure is still elevated under the above circumstances greater improvement may be expected from bleeding than if the blood pressure is low. In fact, when a state of shock is present as in acute coronary thrombosis it may even be harmful. If the onset of hepatic engorgement has been recent and acute as occurs shortly after the development of auricular fibrillation a definite diminution in the size of the liver may result from bleeding. When the liver has remained congested and enlarged for months or years the secondary cirrhotic changes that take place prevent such a decrease from occurring after bleeding.



A phlebotomy should be carried out very rapidly. As large a needle as possible should be used and the entire amount of blood (400 to 700 c.c. of blood, depending on the size of the patient) ought to be withdrawn in about ten minutes. The exact mechanism of the improvement that occurs is not clear. Whether it takes place because of a diminution of the work of the heart by decreasing the volume of blood or by increasing the "tonus" of the heart and thereby its contraction are matters that need not be taken up here. In so far as it may be due to the latter it becomes desirable for the venous blood to be removed rapidly so that the dilated right side of the heart may decrease in size and regain a better tone before further blood returns from the periphery to redilate these chambers. That this mechanism cannot be the sole one involved is illustrated by the beneficial results that occur from bleeding when acute pulmonary edema takes place in a patient with hypertension and sudden left ventricular failure. Here there is no engorgement of the liver or appreciable venous engorgement. This beneficial effect is probably the result of decreasing the return flow to the right ventricle and, therefore, the output to the lungs. This enables a normal balance between the two ventricles to be established. There obviously remain some important questions concerning this matter that need investigation in order that we may have more accurate indications and contraindications for the use of this valuable method of treatment.

Recently instead of blood-letting some have practiced the use of tourniquets on the four extremities. These are applied so that the venous but not the arterial flow is impeded. By this means blood is pooled temporarily in the periphery and prevented from returning to the heart.

After several weeks of rest treatment, a patient such as we have described may be permitted to increase his activities. At first he is allowed to sit in a chair for fifteen minutes forenoon and afternoon if he had been confined to bed. The length of time is gradually increased taking at least one week before he becomes completely ambulatory. When it is planned for him to return to his duties, it is well to have him begin by working only half a day. Some restrictions in his activities must be enforced, as it is apparent that his circulation was not sufficient for the former expenditure of energy. A conference between the physician, the patient and his family will help in ascertaining what part of his work should be curtailed, which entirely given up and which should be retained. It is wiser to have him continue on only one half the previous amount of work with its decreased income, than to try to carry on full duties, knowing that in a short while he will again be bedridden. Medical judgment tempered by good common sense is necessary in giving this advice.

**Quinidine.**—In the preceding discussion of the treatment of patients with congestive heart failure no mention was made of the use of quinidine. This brings up a controversial subject and merits a separate analysis. Quinidine was first proposed as a drug that would change auricular



fibrillation to a normal rhythm. It was accidentally discovered by Wenckebach who was told by one of his patients with auricular fibrillation that he could make his heart beat regularly by taking quinine. It was later learned that quinidine was more effective in producing this change. This was hailed as a great discovery because auricular fibrillation, once established or continuing for a week or more, was practically always expected to persist indefinitely. In fact it used to be called the perpetual arrhythmia. Furthermore, it was also known that heart failure was often precipitated by and dated its onset from the change of the mechanism of the heart beat to auricular fibrillation. It seemed reasonable, therefore, to hope that any drug which could keep the heart regular would be of great benefit. The early reports concerning its use were very optimistic, but, as with many other therapeutic procedures, unfavorable aspects and limitations of its use gradually appeared and now it is known that the former enthusiasm was not altogether warranted. However, there still remains a distinct field for its administration.

In a sense, quinidine is a cardiac poison when given in sufficient doses. It impairs the conduction of impulses and can actually inhibit contractions. It lengthens the Q-T interval of the electrocardiogram and produces slight notching of the T wave. The maximum effect in human beings occurs two to three hours after its oral administration. The pharmacological action upon which it depends, in changing an irregular beat into a regular one, consists essentially of two factors. It lengthens the refractory period of heart muscle and it also slows the speed of the cardiac impulse. It will be recalled that auricular flutter and fibrillation are due to a circus movement of an impulse in the auricles. This impulse keeps traveling around the vena cava at a very rapid rate (300 to 600), always finding cardiac tissue ahead of it that has already recovered from the refractoriness following the previous impulse and thereby permitting a continuous circus motion to persist indefinitely. If the refractory period of the muscle could be lengthened, the impulse might find tissue still refractory and would stop. The circus might be broken up in this way and this would permit the normal pacemaker with its slow inherent rate, which has been held in abeyance by the rapid rate of the circus, to start functioning. On the other hand, if the rate at which the impulse travels around the circus is slowed or the path it takes is lengthened, it affords a longer time for the cardiac tissue to recover from its refractoriness and this would allow the circus wave to continue, thereby tending to perpetuate the circus. One effect of quinidine (lengthening of the refractory period) would tend to break up auricular fibrillation or flutter and the other (slowing of the impulse) would tend to perpetuate it. When the former predominates a regular rhythm is restored and when the latter effect predominates the arrhythmia persists. This explains the variable results obtained from the use of quinidine.

Let us consider what might be expected from restoring a regular rhythm when auricular fibrillation is present. In estimating the results



of therapy both subjective sensations and objective changes must be carefully differentiated. Many patients will say they feel much better after regularization, when on close analysis it will be found that they no longer are annoyed by palpitation. The irregular heart beat is easily felt and causes not only discomfort but some apprehension. They feel the heart less or not at all when it beats regularly, and believe that there must be great improvement because they know that a normal heart should beat regularly. This does not necessarily mean that the heart is more efficient. The best indication that the circulatory state is actually improved is the degree of dyspnea or the objective signs of congestive heart failure. Patients with auricular fibrillation who have improved after quinidine may owe their recovery to the rest in bed and digitalis that was given during the period of observation. To clearly differentiate the effects that are solely the result of quinidine, one should employ all the other therapeutic measures available for a few weeks and then, when the patient is in as good condition as possible, measure what further improvement may follow regularization. When this is done the results will not always be favorable.

Some years ago I undertook a study of this sort. Inasmuch as dyspnea is the outstanding evidence of congestive failure and the vital capacity of the lungs is the best index of the degree of dyspnea, careful measurements of the vital capacity were made. The vital capacity would increase as the condition improved following bed rest and digitalis therapy. The heart rate would slow as was to be expected. After the condition was stabilized quinidine was given. When the rhythm became regular, the vital capacity of the lungs showed no constant alteration—it was slightly less, the same or slightly greater. This meant that breathlessness was not materially improved by regularization of the heart, if the rate previously could be adequately slowed by digitalis. A heart that is beating irregularly at a rate of 60 to 70 can be about as efficient as if it were beating regularly at a rate of 70 to 80. In fact, a slow irregular rate may maintain a better circulation than a rapid regular one. It was occasionally found that in the presence of stubborn congestive heart failure when the regular rate was 100 or more, improvement might first become manifest only after auricular fibrillation developed. What would happen was that digitalis that was given failed to slow the regular rhythm and when auricular fibrillation began the drug slowed the ventricular rate to 60 or 70 and congestion began to clear. In this sense the irregularity at times is an advantage.

There are, however, some definite advantages of a normal rhythm over auricular fibrillation. The formation of mural thrombi in the auricles with the possible development of emboli is much more likely when fibrillation is present. Also, although the heart rate may be kept slow with digitalis when it is irregular, it accelerates less on effort when regular. To these two factors may be added the subjective relief of palpitation.



Taking everything into consideration, it is more desirable that the heart should beat regularly than irregularly. Quinidine sulfate would, therefore, be a very useful drug if this change could be accomplished without risk. However, there are dangers in its use. Occasionally it causes sudden and unexpected death and it may result in the production of emboli. The exact cause of death is not clearly understood. It may result from an embolus but it would not be sudden under these circumstances. I have had three unexpected fatalities in which postmortem examinations failed to show either emboli or mural thrombi. A direct toxic effect on the heart may possibly account for some fatalities, as it is known that quinidine can inhibit the propagation of impulses. Inhibition of auricular beats with temporary disappearance of the P waves has been observed following quinidine. If the same type of effect is produced in the ventricles it would cause sudden arrest of the heart. Another cause of death is respiratory failure. In cats, this, rather than heart failure, is the primary cause of death from toxic doses of quinidine. In fact, when respiration has ceased and the heart is still beating, recovery can result if artificial respiration is instituted and caffeine is administered intravenously even after a lethal dose of quinidine is given to the animal. These experiments showed that the respiratory mechanism can fail while the heart is still viable and if respirations can be stimulated or maintained during the critical period for a long enough time, recovery may take place. Such observations have a direct clinical application, for it follows that caffeine in large doses and artificial respiration may prove effective in tiding over some patients that manifest toxic action from quinidine.

During the early years following the introduction of quinidine it was advised that in order to avoid unfavorable effects, patients with auricular fibrillation to be given this drug should be selected according to certain definite criteria. It was thought that the most suitable were those in whom there was no great amount of enlargement of the heart, the irregularity was of short duration and there was no significant heart failure. In other words, those with the least cardiac disability were regarded as the most favorable. Unfortunately one of the most disastrous results in my own experience occurred when all these favorable factors were present. This woman, about thirty-five years old, had a well-compensated mitral stenosis and had been able to work steadily. When the patient was first seen her heart was regular and three weeks later auricular fibrillation was found. She came into the hospital for a tonsillectomy and it was decided that the heart should be regularized before the operation. She was in very good condition and after a preliminary course of digitalis, quinidine was given. The first day two doses of 0.2 gram (3 grains) were administered. The second day the patient received 0.3 gram (5 grains) three times. That evening the heart was regular, but there quickly developed marked breathlessness and the patient died in several hours. I thought she had a pulmonary embolism from a thrombus in the right



auricle. Postmortem examination showed mitral stenosis and neither pulmonary infarct nor intramural cardiac thrombosis. It was after this experience that the animal experiments on the mechanism of death from quinidine were carried out. Retrospectively it seems highly probable that this patient died of respiratory paralysis and that the same procedure that enabled the animals to survive (*i.e.*, artificial respiration and caffeine) might have saved her life.

Apart from the fatalities due either to cardiac or respiratory effects, emboli may become dislodged as the auricles stop fibrillating and start contracting regularly. There is no known method of predicting in which case auricular thrombi are present and whether emboli will occur. When they do take place, embolic phenomena will develop within a few hours or days after the transition to a normal mechanism. The emboli are more often arterial than pulmonary and may affect almost any part of the body. Hemiplegia is the most common disabling complication. Although such emboli occur in patients with auricular fibrillation who do not receive quinidine, they are much more common during this treatment and must then be regarded as the direct result of quinidine administration.

Another limitation in the use of quinidine when organic heart disease is present is that once a normal rhythm is established, in many cases after a short time there is reversion to the auricular fibrillation that existed before. The regular rhythm is often maintained only for a few days or weeks and then the entire process of regularization needs to be repeated. In many, it appears to be impossible or too difficult to keep the heart beating regularly. Finally in only about 50 to 75 per cent of the cases will quinidine be effective in restoring the heart to a normal rhythm.

Notwithstanding the hazards and limitations of quinidine it occupies an important place in therapy. There are occasional instances in which it alone has been responsible for restoring compensation and one might even say in saving life. The pros and cons must be weighed carefully and with increasing experience a proper selection of those cases in which its use is justifiable will result. In the first place, from a review of my own experience and that of others, it seems to be safe and effective when given to that group of fibrillators, by no means small, who have no organic heart disease. There is a considerable number who have this arrhythmia without other evidence of heart disease. Here it may be expected that practically 100 per cent of the patients will promptly revert to a normal rhythm with only beneficial results. As a corollary of this, it is very effective in the small number who continue to show auricular fibrillation two weeks or so after a subtotal thyroidectomy for hyperthyroidism when the basal metabolic rate has returned to normal. It is useless to give quinidine before operation in such cases or even after operation if the basal metabolic rate has not been effectively lowered by the operation. It is best to wait about two to three weeks after the operation, because in many cases spontaneous change to a regular beat will take place during



this time. In fact, if the change does not take place, careful search for an undetected mitral stenosis should be made.

The main problem concerns the use of quinidine when mitral stenosis, hypertensive or myocardial disease is present. This comprises the group in which fatalities and emboli occur. At the outset it must be appreciated that we have no certain method of avoiding these accidents. The very early cases cannot be regarded as free from hazard, nor can the advanced cases be given up as hopeless. There are some general principles that may guide us in these decisions. All the disastrous results in my own experience have occurred in patients with mitral stenosis. Although serious complications have been reported in non-valvular cases, they are not so numerous. I, therefore, have greater hesitancy in advising it in the former group. When a patient can be restored to a favorable state of compensation on digitalis and the ordinary methods of treatment, it is doubtful whether quinidine should be used. This is particularly true if mitral stenosis is present and the ventricular rate can be kept around 70. On the other hand, if the patient is doing poorly and it seems certain that he will not become ambulatory one would be justified in hazarding a course of quinidine. Occasionally in cases in which there was apparently no hope, improvement has occurred. Furthermore, when palpitation from the rapid irregular beat is a major and disturbing complaint the drug may be tried. In some cases it is evident that as long as the heart was beating regularly, the patient was in comparatively good health and incapacitation dated from the onset of fibrillation. When this disability has been present only a short while, one might risk quinidine therapy in the hope of preventing the slow downhill course that often follows the development of auricular fibrillation.

Whenever it is decided to use quinidine for persistent auricular fibrillation, the patient should be treated in a hospital unless circumstances do not permit it. The advantage of hospitalization is that changes in the mechanism of the beat may be more readily followed if electrocardiograms can be made whenever needed. Ordinary methods of treatment including digitalis should be employed until as much improvement as possible is thereby obtained. A maintenance dose of digitalis is continued during the period of quinidine treatment. The exact amounts of quinidine that are employed and the speed with which the dose will be increased will vary with the urgency of the circumstances and somewhat with the custom of the physician. The following is a routine course from which one can make individual variations as occasions arise. The first day 0.2 gram (3 grains) are given twice at four-hour intervals. This is a trial dose and is supposed to test the patient for peculiar susceptibility to the drug. There are rare individuals who react poorly even to small doses of quinine or its allied preparations and develop a rash, ringing in the ears, nausea, vomiting, diarrhea, faint feeling or syncope. When no untoward effects are produced, the dose is increased on the second day to 0.3 gram (5 grains) three times a day, on the third day to 0.4 gram



three times a day, the fourth 0.5 gram three times a day, etc., increasing 0.1 gram each day until the desired result occurs or the drug has to be discontinued. Another method is to increase the dose 0.1 gram each dose instead of each day.

During this treatment the heart should be examined just before each dose is given to see if regularization has occurred. If the rhythm becomes regular the dose is dropped to 0.2 or 0.3 gram three times a day in the hope that the rhythm will remain regular. Often these comparatively small doses are sufficient to prevent the resumption of auricular fibrillation. In fact, in some cases the rhythm continues to be regular without any further medication. Whether a constant dose of quinidine will be necessary can only be determined by trial and error. Occasionally it may require large daily doses to procure the desired results, although generally if the smaller doses do not suffice, it will be better to accept the arrhythmia as permanent and discontinue quinidine entirely. I recall an instance in which 0.8 gram three or four times a day was necessary to restore the heart to a normal rhythm and that same daily dose to prevent a return of the arrhythmia. This was continued for months and enabled the patient to work, whereas otherwise he would have been bedridden with congestive failure.

During the early days when increasing doses of quinidine are given the ventricular rate often rises while the rate of impulse formation in the auricles slows. The acceleration of the ventricles is undesirable and palpitation becomes more uncomfortable. This temporary aggravation of the condition is to be expected but must not be allowed to last too long. In several days it must be determined whether the rhythm will revert to normal or not. The drug should be omitted if any untoward toxic effects, especially syncope or marked acceleration in rate, develop. When it is found that regularization does not occur, quinidine should be stopped entirely. It should not be given in small doses, like digitalis, for long periods of time, because it will fail to restore a regular beat and only make it more difficult for the digitalis, which the patient is receiving, to keep the ventricular rate slow. In other words, quinidine is given to make the heart regular or to keep it so, but not for any length of time if auricular fibrillation persists.

There is some reason to believe that quinidine might be useful in the prevention of auricular fibrillation in those prone to develop this irregularity. One might expect that the hazards of its use would be avoided or at least diminished if it were given to some patients before fibrillation develops. The difficulty is that there is no known method of predicting when this irregularity will occur and so one can rarely be convinced that the treatment is accomplishing its purpose. Nevertheless, I occasionally advise patients with well-marked mitral stenosis who are well compensated to take 0.2 gram quinidine sulfate two or three times a day indefinitely, in the hope that this distressing type of irregularity may be prevented.



Quinidine has other uses than in the treatment of permanent auricular fibrillation. It has been valuable in various forms of paroxysmal rapid heart action in preventing recurrences. For this purpose it needs to be taken daily for long periods of time. It is very effective in controlling ventricular tachycardia and often inhibits the occurrence of extrasystoles of various forms. These therapeutic problems have been taken up previously in other chapters and need not be considered here. Suffice it to recall that it has an important place in the treatment of many arrhythmias but should be avoided whenever there are defects in conduction.

**Surgical Procedures.**—The inadequacy of medical treatment for patients with chronic heart disease, just as in other fields of medicine, has impelled the profession to seek further aid from surgery. The heart has been the last important organ to enter the scope of therapeutic surgery. It is evident that operative work on the human heart will necessarily be difficult. The circulation cannot be arrested for more than a brief time without sacrificing the life of the body even if the heart beat can be restored. The brain in particular does not withstand anoxemia for more than several minutes. Until a satisfactory artificial circulation is developed that will nourish the systemic organs and even the coronary arteries, any lengthy operative procedures on the inside of the heart will be difficult or impossible. This is just as important as an apparatus for artificial respiration was for the development of pulmonary surgery. Despite these handicaps, heroic attempts now and then are made to explore this new field. Traumatic wounds of the heart are now successfully sutured. Considerable progress in pericardial surgery has been made (Chapter 5). Indirect methods of beneficially affecting the heart have been applied by altering the nervous system. Cervical sympathectomy, dorsal ganglionectomy, and paravertebral alcohol injections of the dorsal rami and ganglia have been employed with some success in the treatment of angina pectoris (Chapter 6). Daring attempts at incising and enlarging the orifice of a stenosed mitral valve have also been made. When the first case of this sort was reported in 1928 by Dr. E. C. Cutler and myself, because the patient survived and seemed somewhat better as a result of the operation, we were hopeful that the operation of valvulotomy might prove useful. The extremely high mortality in subsequent cases quickly placed this operation in disrepute.

A simpler operation has been tried on rare occasions to give relief, when there is marked enlargement of the heart, *i.e.*, decompression of the chest. Occasionally this has been done believing that pericardial adhesions are present only to find subsequently that the pericardium was normal. Despite this mistaken diagnosis, clinical improvement has been noted in some such cases. I had one experience in which the removal of a generous portion of several ribs overlying the precordium was of considerable benefit. This patient was about twenty-five years old and had mitral stenosis, auricular fibrillation, and marked cardiac enlargement. The circulation was maintained in a fair state of compensation



with the constant use of digitalis. I had followed this case for many years. The patient finally developed dysphagia which resulted from pressure of the enlarged left auricle on the esophagus. This was purely a mechanical difficulty and did not respond to ordinary methods of treatment. The dysphagia was promptly cured by removing, under local anesthesia, portions of ribs overlying the precordium. Not only was the patient able to swallow normally after this operation, but palpitation, which had been very annoying before, was much improved. She then did not feel the rapid irregular heart beat, because it was no longer pounding against bony ribs, but rather on a soft cushion of muscles and subcutaneous tissue. Even the ventricular rapidity seemed to be better controlled by digitalis than formerly. The patient lived for about nine years after the operation. Such decompression of the chest may be indicated whenever an enlarged heart is producing distressing symptoms as a result of pressure. This may not only involve the esophagus but also the left bronchus causing harassing cough. Enlargement of the left auricle and of the pulmonary artery may also produce hoarseness or aphonia as a result of paralysis of the left vocal cord caused by pressure on the left recurrent laryngeal nerve. Similar operative procedures that decompress the chest may possibly prove useful in such conditions.

Finally, the hopelessness of many cases of advanced cardiac disease has been responsible for the development of the recent surgical procedure, *i.e.*, complete thyroidectomy. Its application in the treatment of patients with angina pectoris has already been discussed (Chapter 6). It also has been performed for intractable congestive heart failure, due either to valvular or myocardial disease. It was known that when the basal metabolic rate is elevated as in hyperthyroidism, and there is congestive heart failure, a subtotal thyroidectomy with a subsequent fall in the metabolic rate caused the heart failure to disappear. It was also known that in myxedema associated with a sluggish circulation, congestive heart failure was very rare. It was, therefore, theoretically assumed that by producing myxedema in a patient without hyperthyroidism who has heart failure the demand on the heart might thereby be lessened to meet the supply. However, it was overlooked that in the production of myxedema, not only is the demand diminished by lowering the metabolic rate, but the supply is also depressed, for the volume output of the heart is diminished and the velocity of blood flow is slowed in spontaneous and artificial myxedema. If the circulation is improved by this procedure, therefore, there must be other factors at work. The inherent metabolism of the heart may be altered so that it does its necessary work more efficiently and with less proportionate expenditure of energy. Another possibility is that with a partial myxedema, the heart is less sensitive to certain reflexes or internal hormones such as epinephrine. Finally a most important factor is the size of the heart. A certain amount of dilation of the heart may be beneficial, but excessive dilation impairs the efficiency of the circulation. In congestive heart failure the heart is al-



ready dilated to a greater or lesser extent. When myxedema is produced, there is a tendency for further dilation to occur. May not improvement depend on this unpredictable factor, *i.e.*, whether the further dilation of the heart is excessive or not? At any rate the pathological physiology of this problem is still unsettled.

The clinical results of complete thyroidectomy have been variable. In those suffering from extremely advanced lesions, if improvement occurs, it may not last long enough to warrant the operation. On the other hand, the operation does not seem justified if the disease is only slight or moderate or if the disability is not great, because of the handicaps attending partial myxedema. There remains a small group of cardiacs, neither too sick nor too well, who may be suitable for this operation. There is no doubt that some of the patients who had complete thyroidectomy have been improved as far as their symptoms of heart failure are concerned, but it is hoped that a simpler method might be devised to obtain similar results without producing the ill-effects that follow the removal of an important vital organ. This work must still be regarded as in the experimental stage, but has already served as a forceful stimulus to the exploration of new fields that might bring relief to those suffering from intractable heart disease. At present I do not advise total thyroidectomy in the treatment of persons with chronic cardiac disease.

Another surgical procedure that is being employed for hypertension and to some extent for hypertensive heart failure is sympathectomy. Various types of operations are being tried, but it appears that the dorso-lumbar sympathectomy of Smithwick is at present most promising. More extensive division of the sympathetic nervous system, including all the dorsal and lumbar branches, may prove to be more effective. Already many successful results have been obtained, even when myocardial involvement was present as shown by the presence of gallop rhythm and markedly abnormal electrocardiograms. This approach to the problem of hypertension and its complications deserves our most careful interest and attention.

There is one peculiar though rare form of congestive heart failure that responds most dramatically to surgical treatment, *i.e.*, the type following *arteriovenous aneurysm or fistula*. As a result of a traumatic wound, or less frequently following an infection, a communication may become established between an artery and an adjacent vein. Blood is then shunted through the circulation and the work of the heart becomes definitely increased. In the course of time dilatation of the heart, murmurs and congestive failure are apt to develop if the blood vessels involved are of significant size. The diagnosis is easily made by the history and the findings of a continuous murmur and thrill at the site of the aneurysm with accentuation during systole. Furthermore, compressing the fistula produces an immediate and characteristic slowing of the heart rate. The surgical obliteration of the communication between the artery and vein



can result in a complete disappearance of all subjective and objective evidence of cardiac failure.

We have already witnessed some important results from the great interest displayed, especially in America, in cardiac surgery. No doubt some efforts will prove in vain, but progress can be attained only if attempts are made. One recent development mentioned in Chapter 11 is the successful ligation of patent ductus arteriosus. Let us hope that much more surgical progress in the treatment of chronic heart disease will be made in the near future.



## 21

### CLINICAL ELECTROCARDIOGRAPHY

#### INTRODUCTION

ELECTROCARDIOGRAPHY has become an essential part of our methods of examination of the heart. By its use the diagnosis, prognosis and treatment of heart disease have been considerably improved. At present, conditions can be recognized which were entirely beyond the scope of the most experienced physician a generation ago and treatment can be more intelligently directed. This does not mean that the older and simple methods of clinical examination have lost any of their usefulness. An able clinician who knows nothing about the string galvanometer can still do better work than an expert in electrocardiography who has limited bedside experience and inadequate clinical judgment.

The information obtained by this method must be appraised carefully and used as part of a chain of data concerning the problem involved. This is true of many laboratory procedures. At times a negative finding does not eliminate a suspected diagnosis and likewise a positive finding may have nothing to do with the major complaint. In the early diagnosis of osteomyelitis, the *x*-ray may show no pathological change; on the other hand, finding diverticula in the intestinal tract on *x*-ray examination may be entirely irrelevant, when the symptoms are actually due to a carcinoma of the bowel. Similarly, normal electrocardiograms may be obtained in patients suffering from the most serious types of heart disease such as bacterial endocarditis and angina pectoris. Furthermore, at times when abnormal electrocardiograms are found, care must be exercised in interpreting them in terms of the patient's illness. Such changes in the heart may be incidental and the primary disease, for example, may



be gallstones or a ruptured peptic ulcer rather than heart disease. Finally, as often occurs when a new procedure is introduced, undue emphasis is placed on and incorrect deductions are made from the information thereby elicited. As will be seen in the following pages, many patients who have shown changes in the electrocardiograms which formerly were regarded as very grave, have continued to progress favorably for years and years, requiring us to revise our views concerning prognosis. It, therefore, must be clear that the knowledge derived from electrocardiography, although important, is limited in its scope and needs careful interpretation.

There are several main purposes in the study of clinical electrocardiography. Of first importance is that with a clear understanding of this subject it becomes less and less necessary for the physician to depend upon it. This sounds paradoxical, but experience shows that the more one knows about electrocardiography, the more one can predict what tracings will show under given circumstances, so that those who are most familiar with the subject need it the least. When it was first introduced into clinical use, it quickly clarified all the irregularities of the heart beat. It has taught us how to recognize most of them at the bedside and, when in doubt, it serves as a final check in deciphering disturbances in the mechanism of the beat. It is no longer sufficient for a physician to say that a heart is "slightly irregular" or "less irregular" unless he adds the type of irregularity. More recently it has thrown a great deal of light on disease of the coronary arteries and myocardial infarction. This information can be of tremendous importance for there may be no other way of ascertaining it. Occasionally the electrocardiogram aids in the more accurate diagnosis of valvular disease. Finally, there are certain conditions in which it is indispensable in directing proper drug therapy. The time has come then, when all physicians, even those who will never be directly engaged in the technical work, should have some understanding of this subject.

**Principles of the Electrocardiogram.**—Although the apparatus used in electrocardiography is very complicated, the principles underlying it are very simple. It has long been known by physicists that if an electrical conductor is placed in a magnetic field and a current is passed through this conductor, it will move perpendicularly to the lines of force of the magnetic field. It will move in one direction or another depending on whether the current passes down or up this conductor. The amplitude of this movement will depend on the strength of the magnetic field, the magnitude of the current going through the conductor and on its tension (whether it is loose or tight). The human heart, like all contracting muscles, produces an electric current (an electromotive force). This spreads throughout the body which is also a conductor and it only becomes necessary to deliver this current to the physical apparatus in order to record it. The apparatus consists of an electromagnet with its north and south poles very close together thereby producing a concentrated



magnetic field in its gap. Between these two poles is suspended the conductor, which consists of a very finely drawn out quartz string (about 0.004 mm. in width) which has been covered with a metal, like silver or platinum. An electric source of light and condenser are used to illuminate the movements of the string and a microscopic arrangement to magnify the movements approximately 500 fold. A timing device interrupts the light at fixed intervals thereby permitting accurate measurements of the time relations of various events. Finally the shadow of the string is photographed by a camera.

To deliver the current from the heart to the string galvanometer, electrodes are placed over various parts of the body and are connected with the two ends of the sensitive string in the galvanometer. There are other currents made by the body which deflect the string to one side or another, but inasmuch as they are more constant in their behavior, an equal and opposite current is sent in from a simple dry cell attached to the machine which neutralizes all extraneous currents and restores the string to its center. Now we are left with the only appreciable changing current of the body, *i.e.*, that made by the heart. A final important step in the technique is the standardization of the curves. The string is loosened or tightened with each tracing so that when 1 millivolt of current is passed through it the shadow will deflect 1 centimeter. In this way curves obtained from a patient at one place are the same as those taken on the same patient at any other place, provided the state of the heart does not change. In fact, the human electrocardiogram, although different in different people, is almost as distinctive as finger prints and remains unchanged over long periods of time unless altered by disease or some other extraneous cause.

It has been stated that electrodes are placed on various parts of the body to take off the currents from the heart. It is obvious that two points are needed to complete the circuit and it has become the accepted method to use certain given positions in general practice. The resultant electrical change and, therefore, the amount and direction of the movement of the string, will vary depending on which two points are taken. It will be greater if the line connecting these two points is more parallel to the axis of the electrical change in the heart at that moment and will be less if it is more perpendicular to it. In this way evidence can be obtained concerning the electrical axis of the heart, a discussion of which will be taken up later. The accepted points of application of the electrode for clinical use are the two forearms and the left lower leg. When the right arm and left arm are used it is called Lead I; when the right arm and left leg it is Lead II; when the left arm and left leg it is Lead III. More recently a fourth or chest lead has been employed in which the exploring electrode is placed over the precordium in the region of the apex, near the left sternal border or at some other point over the heart and the indifferent electrode is placed on one of the limbs or the back. In each in-



stance a separate and different tracing is obtained when any two of these points are led off to the electrocardiograph machine.

Another type of apparatus that has come into common use is an amplifier tube electrocardiograph. Here the minute currents from the heart are greatly amplified and the recording mechanism is a moving mirror that reflects a beam of light. A very large resistance is included in the system which does away with overshooting. Furthermore, extraneous currents that are essentially constant in magnitude, such as those coming from the skin, do not interfere with the recording of the electrocardiograph, as the base line automatically returns to its resting level. Thus, physicians are enabled to use a small, portable and sturdy machine that is satisfactory for general practice.

**The Normal Electrocardiogram.**—Normally the impulse that initiates the heart beat arises at the sino-auricular node of Keith-Flack, which is

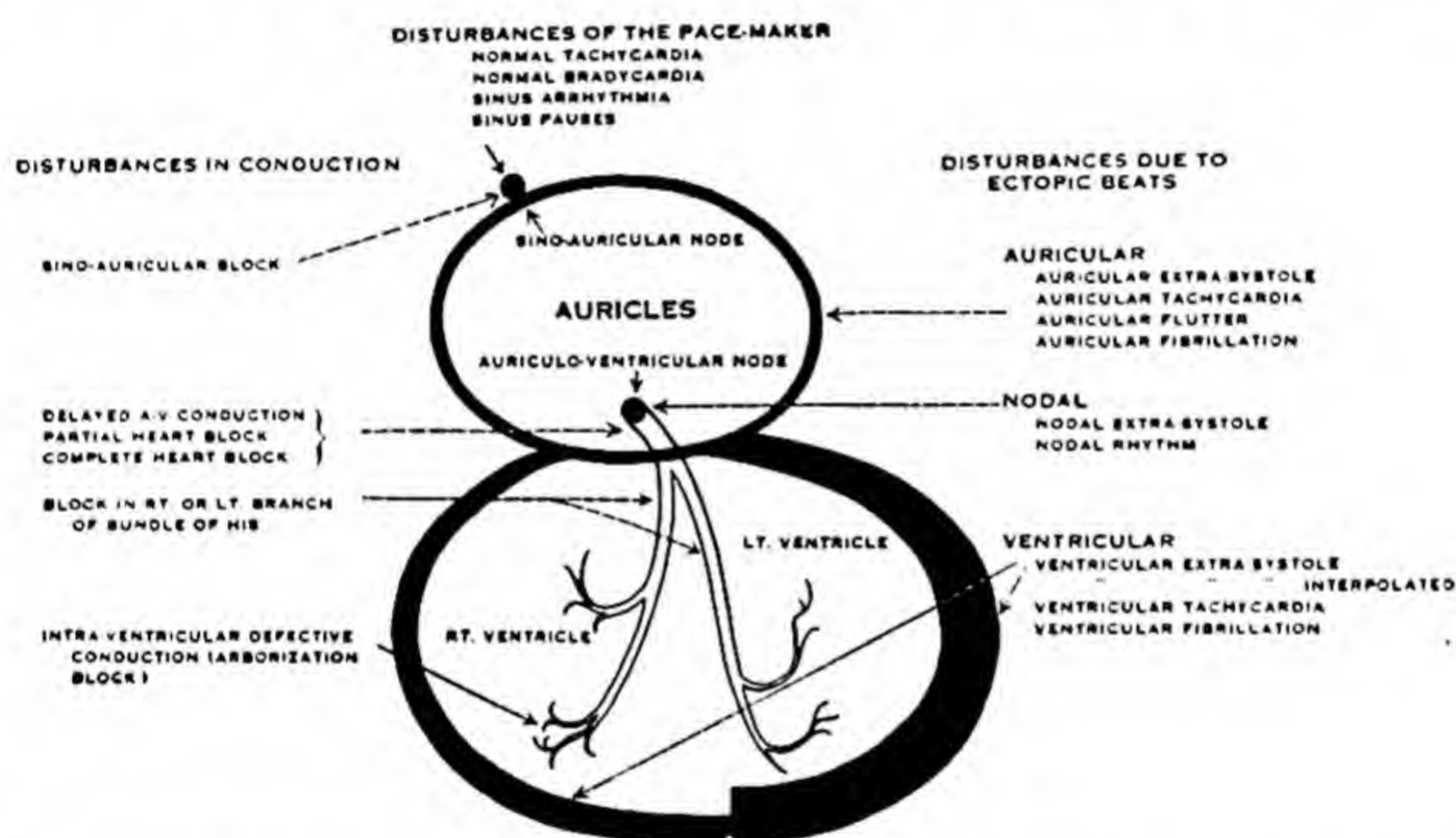


Fig. 1.—A schematic diagram illustrating the common types of disturbances in the mechanism of the heart beat. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

a specialized bit of tissue at the junction of the superior vena cava and right auricle. This tissue is different in structure from the rest of the cardiac musculature in that it is rich in nerve fibers and ganglia cells. It has been shown that the wave of excitation starts here and so this node has been called the pacemaker of the heart. This wave spreads rapidly over the auricles at the rate of about 1000 mm. per second and causes both auricles to contract simultaneously. There is no specialized conduction path in the auricles, the spread being through contiguous muscle bundles. The wave of excitation then reaches the junctional tissue between auricles and ventricles. This structure is called the auriculoventricular node of Tawara which continues into the bundle of His. This junctional tissue also has a specialized structure like the sino-auricular node. The node itself lies at the posterior portion of the auriculoventric-



ular septum where the coronary vein empties into the right auricle. It passes forward to the interauricular septum where it becomes the bundle of His and at the top of the interventricular septum this bundle divides into two branches (right and left) extending down each side of the ventricular septum. The specialized conduction path continues with finer and finer arborizations (Purkinje fibers) and spreads throughout the two ventricles, being particularly abundant in the endocardial surface of the

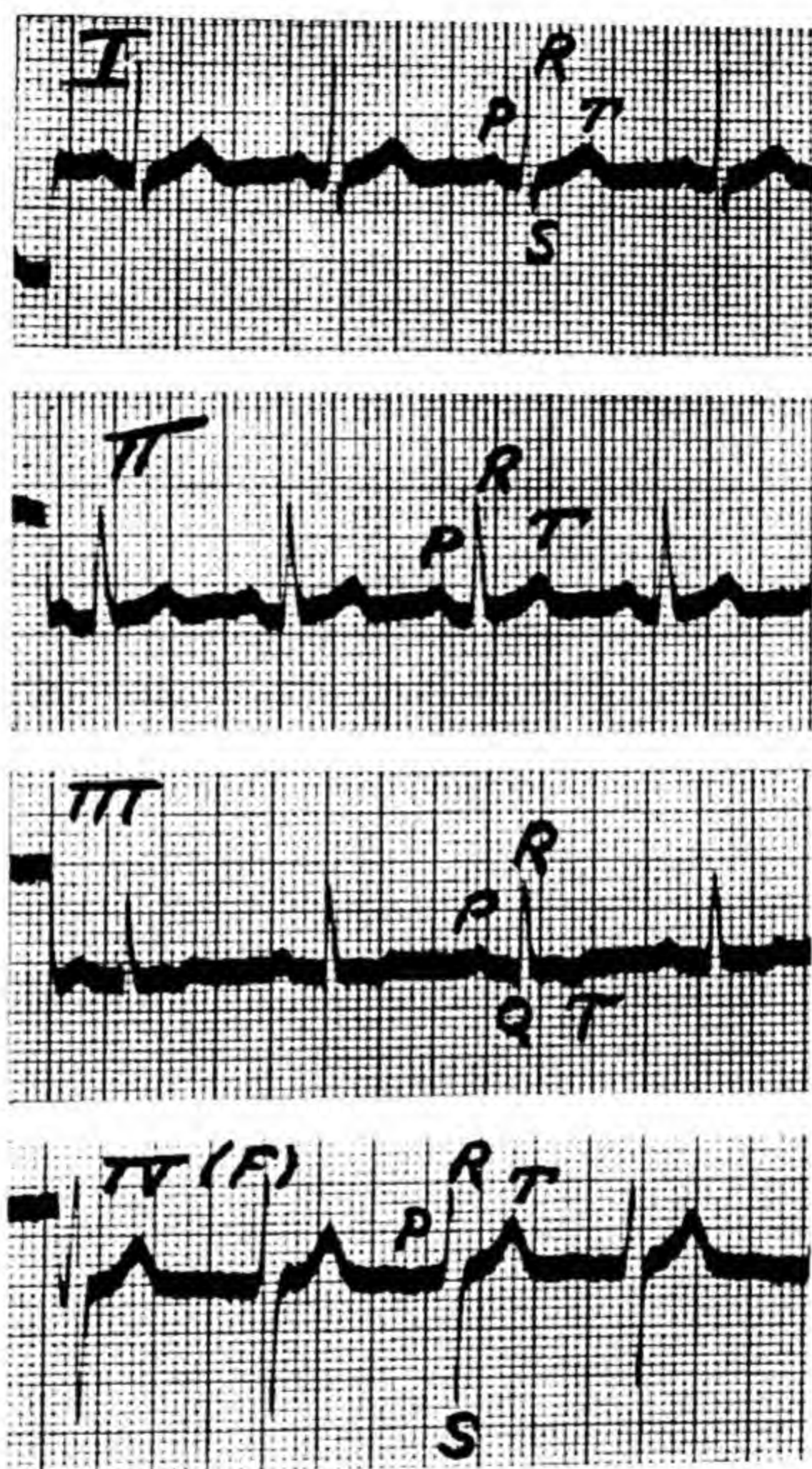


Fig. 2.—Normal Mechanism. The upper three curves show the customary three leads. For the lowest curve (Lead IV F) the exploring electrode is at apex, indifferent electrode on left leg. At the beginning of each tracing the standardization is photographed (1 centimeter = 1 millivolt). P is the auricular complex, QRS is the initial and T the final ventricular complex. The time is indicated in  $\frac{1}{2}$  and  $\frac{1}{5}$  seconds. Note that  $T_1$  may be flat normally.

heart. At the a-v node and the bundle of His the impulse is delayed and then spreads rapidly through the main branches and Purkinje apparatus traveling from the endocardium to the epicardium. The arrangement is such that both ventricles are stimulated to contract simultaneously. It will be helpful in visualizing this sequence of events to refer to Figure 1 which is a schematic picture of how the impulse travels in the heart and how it may be disturbed.



When the electrocardiogram is taken of a normal individual a series of waves will be found. They have been arbitrarily called P, Q, R, S and T waves (Figs. 2, 3, 4 and 5). These electrical complexes will differ in

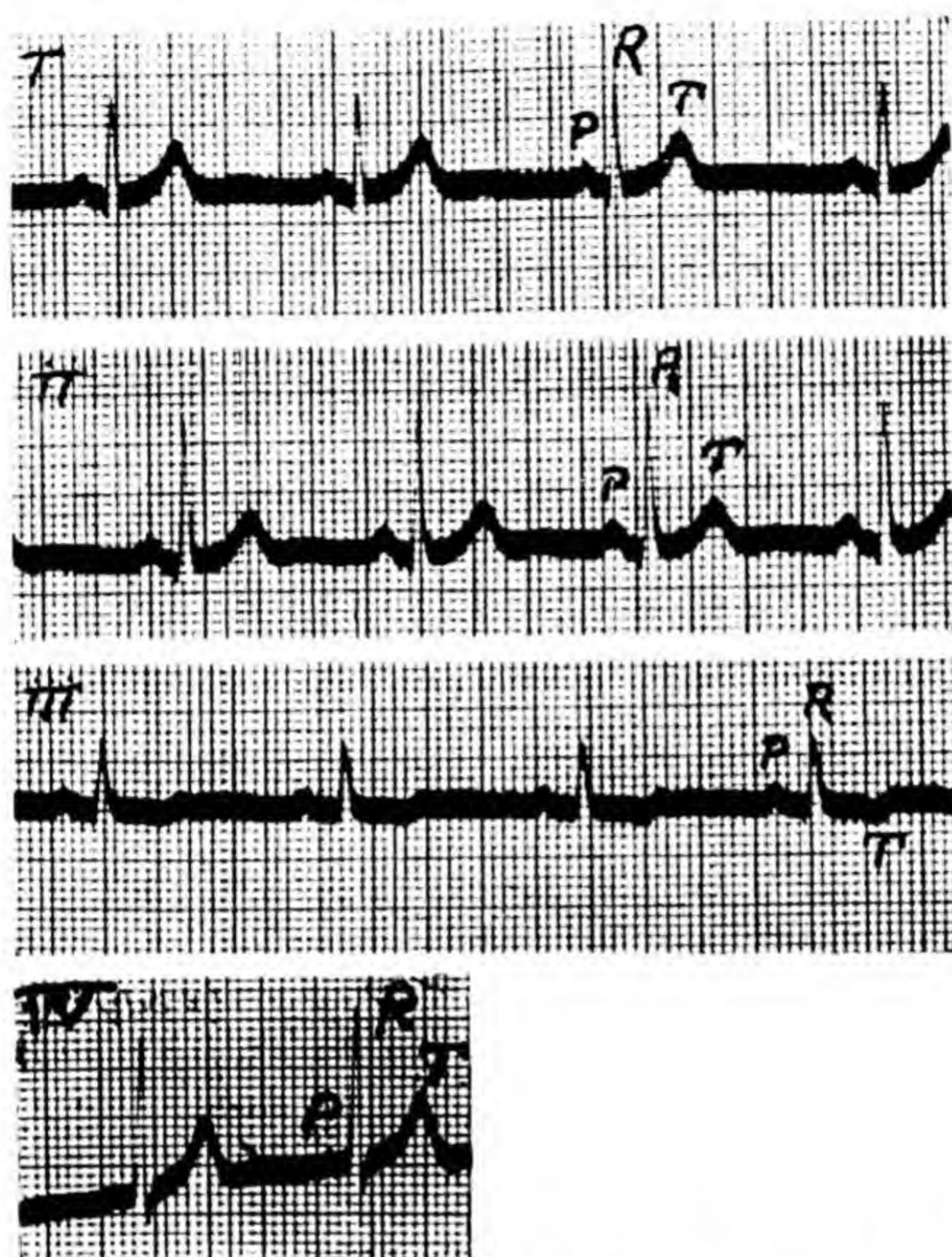


Fig. 3.—Normal Curves. This patient, aged forty-nine, felt well and showed nothing unusual on physical examination and yet seven days later had a typical coronary thrombosis from which he recovered.

form in the various leads but we need not at present be concerned with this aspect of the subject. The P wave represents the electrical disturbances that take place in the auricles. This wave as well as the other

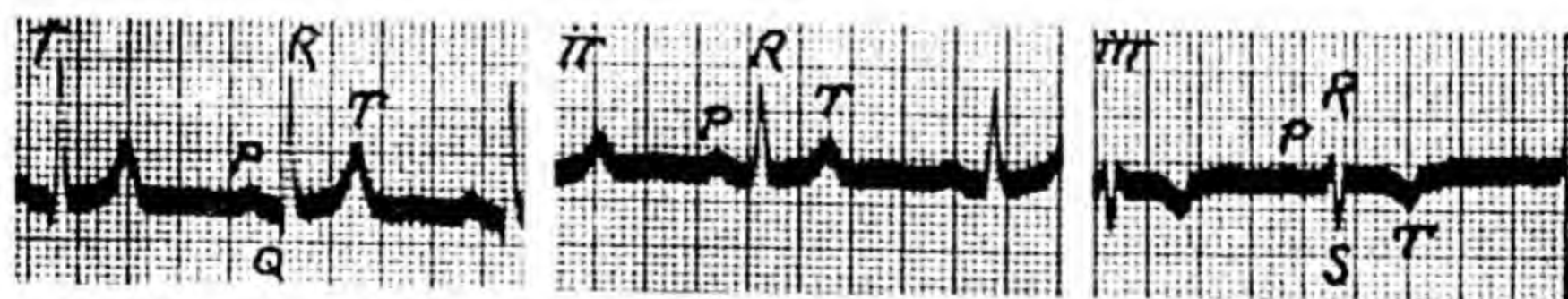


Fig. 4.—Normal Curves. Note the inverted T<sub>1</sub> and the slight left ventricular preponderance which are common findings in normal persons, especially after the age of forty, and in stocky individuals.

important ones are apt to be most prominent in Lead II. All the other waves are due to ventricular activity and can be divided into two portions, the initial deflections (QRS) and the terminal deflection (T). The first downward wave, if followed by an upward deflection, is called a Q



wave. The first upward wave, whether preceded by a downward one or not, is called an R wave. A wave that is directed downward which follows an R or is not followed by an R is called S. If there is a second upward deflection after an S wave, it is called  $R^1$  and similarly a second downward deflection after  $R^1$  would be  $S^1$ . If there is only one deflection and it is downward it may be called QS. There is some controversy about the proper terminology of the initial deflections but the above nomenclature has the sanction of present usage. After the QRS complex there follows a brief iso-electric line which gradually blends into a smooth rounded T wave. Occasionally there may be seen a small U wave after the T which has an obscure origin and no practical significance.

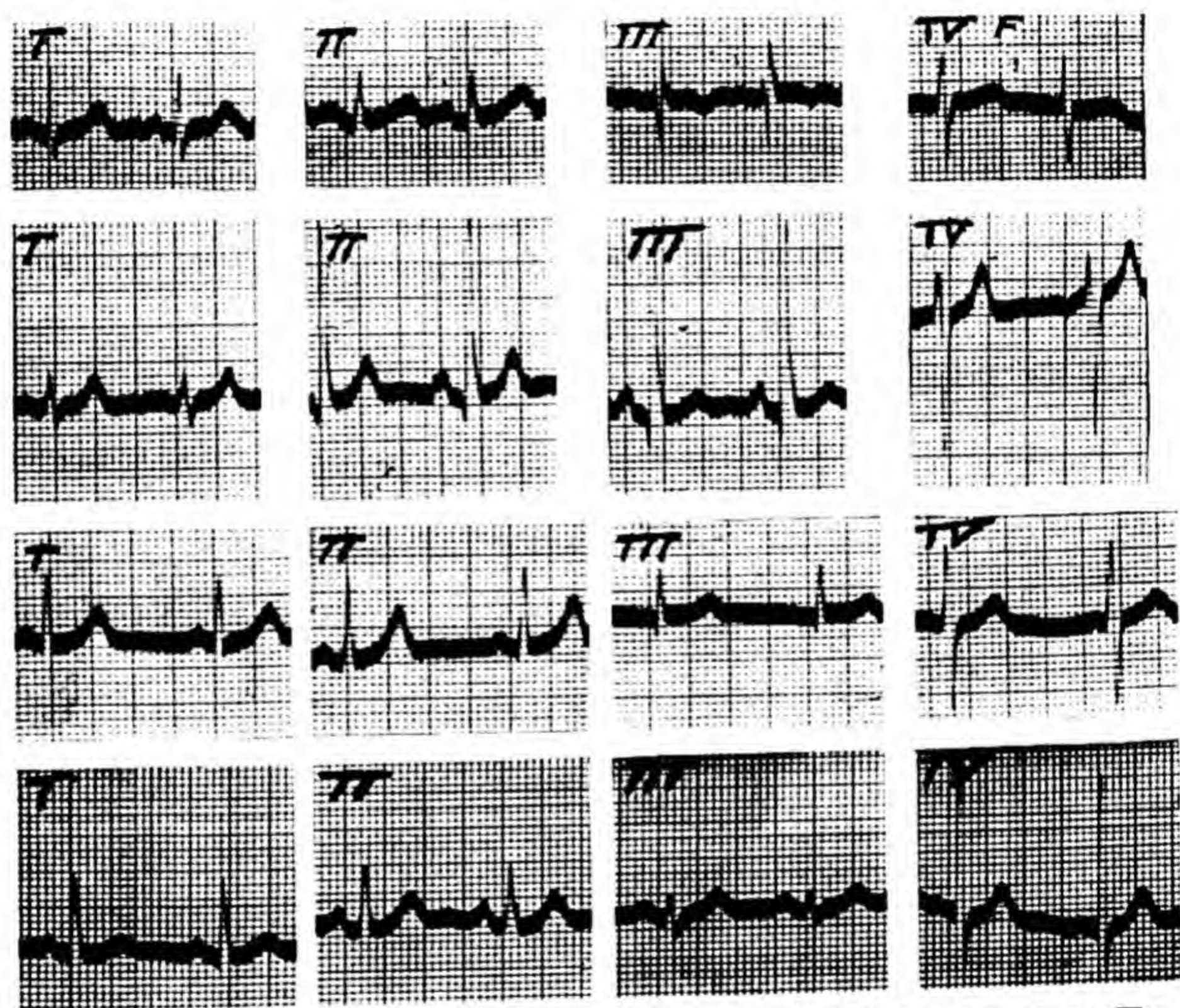


Fig. 5.—Four Sets of Normal Curves. The first is from a child eight years of age. The next three are from adults twenty-three, forty and fifty-three years of age. None had any evidence of heart disease. Note variations in the form of the normal electrocardiograms.

The P wave normally is upright and measures about 0.5 to 2.5 mm. in height. The P-Q or P-R interval which measures the time it takes an impulse to go from auricles to ventricles (conduction time) varies from 0.12 to 0.20 second. Beyond this it is regarded as pathologic. Most of this delay takes place at the a-v node and bundle of His. The normal P-R interval decreases with increasing heart rates and is shorter in children than in adults. In children under six years the range is 0.13 to 0.17 seconds and for those between seven and thirteen years it is 0.14 to 0.18 seconds for rates of 130 to 70 respectively. Normally Q and S waves may or may not be present. The R wave varies in height from about 5



to 15 mm. The T normally is upright in Leads I and II and may be upright or inverted in Lead III. Its amplitude will range from 1 or 2 mm., to 4 or 5 mm. The duration of the QRS is an important part of the study of a tracing. Normally it will be found to be between 0.04 and 0.08 second. When it reaches 0.1 second it is regarded as delayed. The duration of the Q-T interval which is an accurate measurement of the length of ventricular systole is about 0.4 second but will vary with the cardiac rate.

The QRS complex really measures the time it takes the impulse to spread throughout the two ventricles and reflects the integrity of the two branches of the bundle of His. The thickness of ventricular muscle will affect this time to only a slight extent. The T wave or terminal portion of the ventricular complex may be regarded as due to the process opposite to that producing the QRS. The latter is the advance of the electrical or physicochemical process, the former is the retreat; the one contraction, the other relaxation.

Normally the P, R and T waves which are regarded as the more constant deflections should be greatest in Lead II and the sum of the height of the waves in Leads I and III should equal Lead II. Because of the great variations in the character of these complexes in normal individuals, a considerable experience is required to become familiar with the normal and caution must be exercised before slight alterations are regarded as significant.

Recently it has been suggested that during the last two months of pregnancy electrocardiographic evidence of the fetal heart can be obtained by taking a lead from the left arm and right leg of the mother. Very minute waves may be seen regularly interspersed amongst the mother's electrocardiographic tracings. Insertion of electrodes into the rectum or vagina of the pregnant woman may enable one to obtain larger electrocardiograms of the fetal heart.

Because the precordial or fourth lead has only recently been introduced, confusion has arisen as to the proper technique to be used and concerning the criteria for the normal variations of the curves obtained. At first, electrodes were applied which normally resulted in inverted waves in Lead IV. Some used one point over the precordium for the exploring electrode and others used a different point. Likewise there were differences in the site for the indifferent electrode, some using an arm, others a leg or the back. Finally a committee of the American Heart Association and the Cardiac Society of Great Britain and Ireland recommended a technique which is now followed by most physicians. It is suggested that the application be such that normally the waves in Lead IV are upright and thereby comparable to those obtained in the three conventional leads. There are still differences of opinion as to the exact point over the precordium to be used for the exploring electrode.

In taking the ordinary three leads it does not matter whether an electrode is applied an inch higher or lower on one of the limbs. In taking Lead IV, however, moving the electrode 1 inch over the precordium



produces profound alterations in the tracings. In fact, waves may change from an upright to an inverted direction. This will inevitably introduce confusion in comparing precordial tracings obtained on the same patient at different times, unless the exact site of application of the precordial electrode in relation to the position of the heart is controlled. Furthermore, slight differences in the curves result when different points such as the back, arm or leg are used for the indifferent electrode. Finally, for routine use it would be desirable to have only one precordial lead which would afford the most useful information and which would be subject to the least confusion, although under certain circumstances several may be necessary. At present when only one precordial lead is taken it seems best to couple an electrode from the region of the apex of the heart to one on the left leg. Such a lead is generally called  $CF_4$ . Curves obtained in this fashion will show a very small  $P_4$ , generally upright but occasionally inverted. The initial ventricular deflection will be upright ( $R_4$ ) and  $T_4$  will be upright (Figs. 2, 3, 5). In the first years of life  $T_4$  may normally be inverted and occasionally normal adults may show an inverted  $T_4$ . Rarely a lead may be taken from the esophagus to help in the diagnosis of posterior myocardial infarction.

#### DISTURBANCES OF THE PACEMAKER (SINO-AURICULAR NODE)

**Normal Tachycardia.**—It appears that the sino-auricular node has to go through some chemical process in building up material which finally explodes and sends out an impulse. This process normally repeats itself in a fairly orderly fashion at a rate of about 70 per minute. There are numerous common conditions in which the process takes place more rapidly, *i.e.*, exercise, emotion, fever, hyperthyroidism, etc. Under these circumstances the impulse starts in the normal focus and travels across the heart normally but the rate at which it is repeated is increased. This is called *normal tachycardia* or *sinus tachycardia* (Fig. 6). All the complexes have an essentially normal configuration and most of the acceleration takes place at the expense of the diastole of the heart (the T-P interval). Such a condition need not indicate disease of the heart. It was doubtful whether any organic heart disease was present in the patient illustrated by the upper curves of Figure 6 and there certainly was no heart disease in the patient from whom the middle tracings were obtained. At times it is very important to distinguish a normal tachycardia from one due to an ectopic rhythm and it may be necessary to produce vagal stimulation as is illustrated in Figures 30, 35 and 70. When a rapid regular rhythm is due to an abnormal mechanism, vagal stimulation either produces no effect, stops the tachycardia or causes temporary abrupt alterations in the rate, while in normal tachycardia the effect is slight or gradual.

**Normal Bradycardia.**—The vagus and sympathetic control have much to do with the regulation of the rate of impulse formation at the pacemaker. Under certain conditions, either as a result of increased vagal or



diminished accelerator tone, the rate of the heart is unusually slow and sluggish, 45 or less (Fig. 7). This occurs in some normal healthy individuals, particularly tall young athletes, with undernutrition, as a result of jaundice, during sleep and after certain infections. The entire mechanism of the beat is normal and, therefore, the electrocardiogram will be

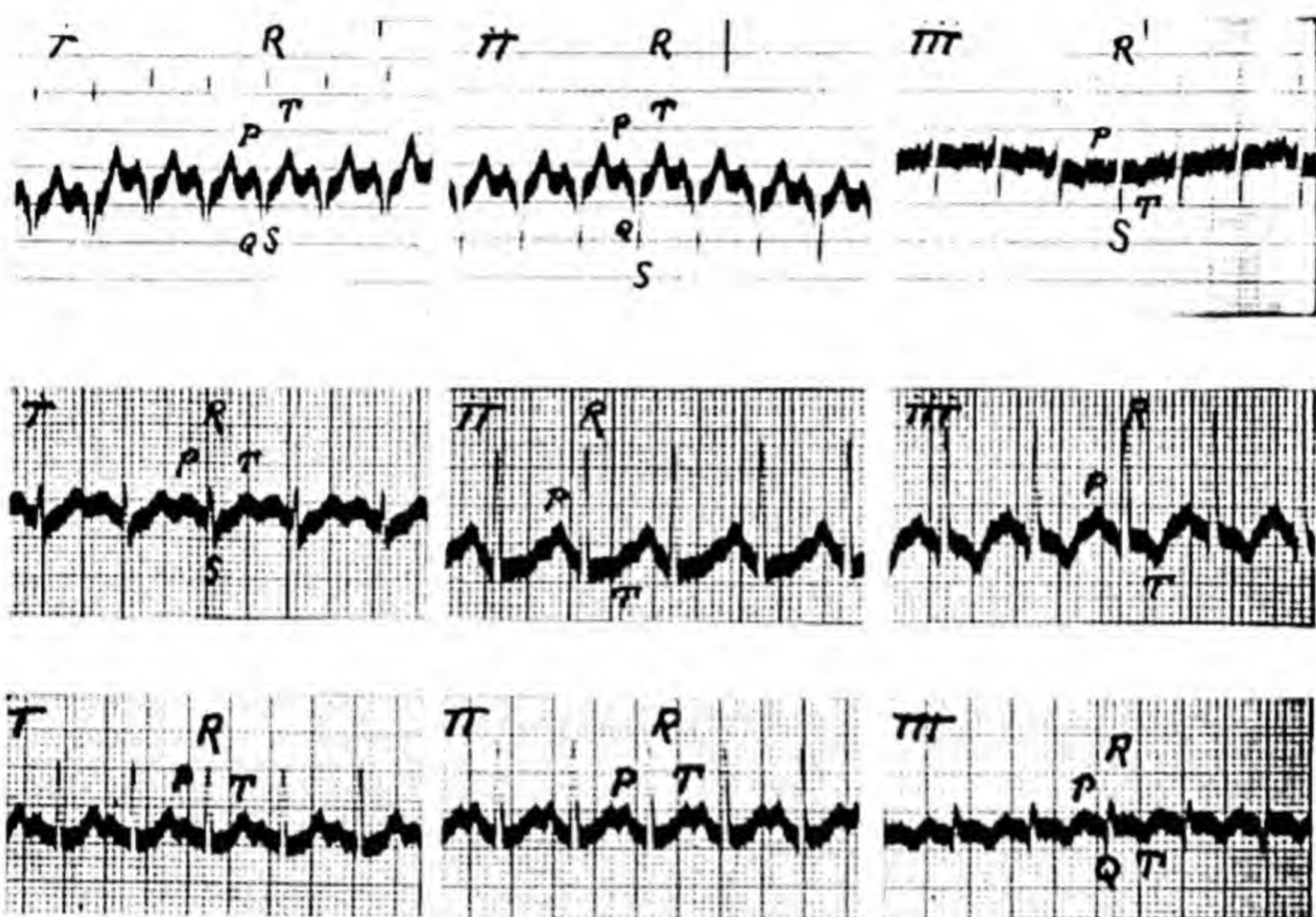


Fig. 6.—Normal Sinus Tachycardia. Upper tracing is from a boy, aged nine, who had a post-scarlatina tachycardia, rate 143. Middle curves are from a boy, aged eighteen, with neurocirculatory asthenia, rate 146. Note all complexes are normal in sequence but merely come at a rapid rate. The  $T_1$  and  $T_2$  in neurocirculatory asthenia are frequently flat or inverted. Lower set is from a boy, aged nine, with acute rheumatic heart disease, rate 166.

normal in every respect except that the diastolic pauses will be great. The condition is called *normal bradycardia*. At times the rate can be below 40 and even 35 with a normal mechanism. At these low levels it must be clearly distinguished from heart block, for with the former the heart is apt to be normal and with the latter the heart is almost always diseased. This can readily be done without special apparatus, for on

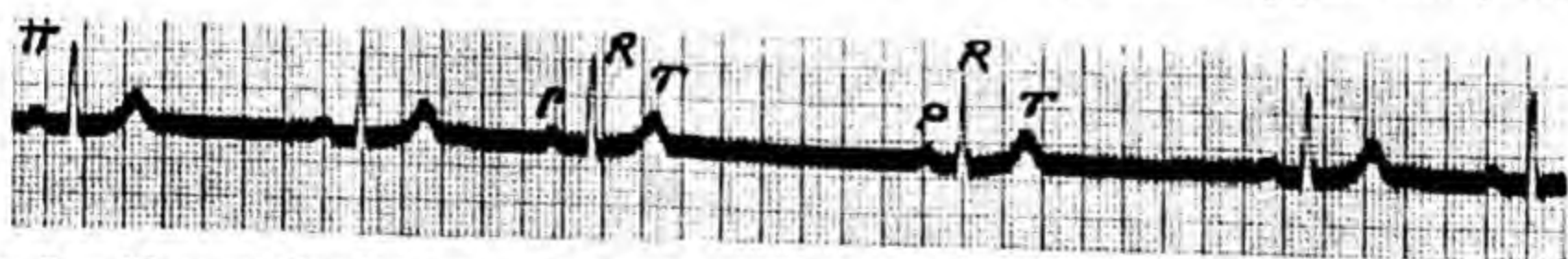


Fig. 7.—Normal Bradycardia. Rate about 42, from a man, aged sixty-eight, who had no symptoms or signs of heart disease. Note that the complexes are normal, the rate is slow and there is a sinus arrhythmia.

exercise the rate will gradually rise to a higher level and then return to the original slow rate if the bradycardia is a normal one; whereas when heart block is present the rate will either change slightly or not at all or sudden interruptions in the length of the heart cycle will be detected.

**Sinus Arrhythmia.**—There is an irregularity of the heart called *sinus arrhythmia* in which gradual acceleration and retardation take place



(Figs. 8 and 9). It is sometimes called respiratory arrhythmia because it often is phasic with respiration. The rate speeds up with inspiration and then slows with expiration. At times it is independent of breathing. The impulse originates in the normal pacemaker and traverses the heart normally, the gradual changes taking place mainly in the length of diastole. Because it is so common in childhood it is also called juvenile arrhythmia. When it is marked or when the changes are abrupt it may be confused with more serious irregularities such as auricular fibrillation or heart block. It occurs in many healthy people, in some older individuals with myocardial disease and after full digitalization. It is apt to disappear entirely if the heart rate is increased artificially by exercise or by diminishing the vagal tone with atropine. The important point is that it must be regarded as an essentially normal phenomenon and does not indicate organic disease. When the heart rate has been rapid and



Fig. 8.—Sinus Arrhythmia. A woman, aged thirty-eight, who had malnutrition but no heart disease. Note gradual increase and decrease of the length of the heart cycles. The changes take place almost entirely in the length of the diastolic pause (T-P interval).

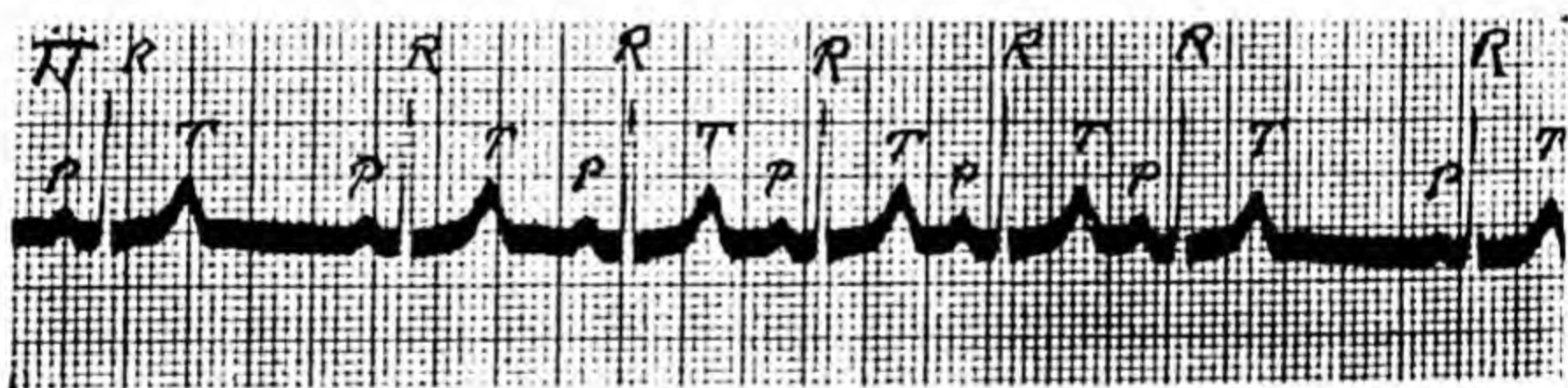


Fig. 9.—Sinus Arrhythmia. From a boy six years of age with a normal heart. Note more abrupt changes in the length of the heart cycles.

regular for some cause, such as hyperthyroidism or rheumatic carditis, the appearance of sinus arrhythmia is apt to indicate that the condition is progressing favorably and the normal vagal control is returning. In fact, it is rare to find this arrhythmia while active thyrotoxicosis or rheumatic carditis exists.

**Sinus Pauses.**—The pacemaker of the heart may become inhibited as a result of certain reflex influences and thereby fail to produce impulses for varying lengths of time. Under such circumstances the whole heart fails to contract and if the pause is sufficiently long, giddiness or actual syncope occurs, presenting clinical features that are similar to those seen in Adams-Stokes disease. This condition is called *sinus pauses* (Fig. 10). The electrocardiographic complexes are normal in form but there appear long diastolic pauses of varying lengths. This might be regarded as an exaggerated form of sinus arrhythmia. The mechanism of this disturb-



ance depends on a reflex stimulation of the vagus such as a vagovagal or carotid sinus reflex. Figure 10 is a tracing of a patient who had spells of unconsciousness precipitated by the act of swallowing or gagging and



Fig. 10.—Sinus Pauses. Note marked irregular slowing of the heart; neither auricle nor ventricle contracts. This resulted from vagus stimulation following gagging (probably a vagovagal reflex). The irregularity of the base line is an artefact and due to movement of the patient. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

accompanied by a peculiar sensation in the throat. It was found that each time a tongue depressor was applied to the tongue to examine the pharynx an attack occurred, during which the heart stopped for several seconds.

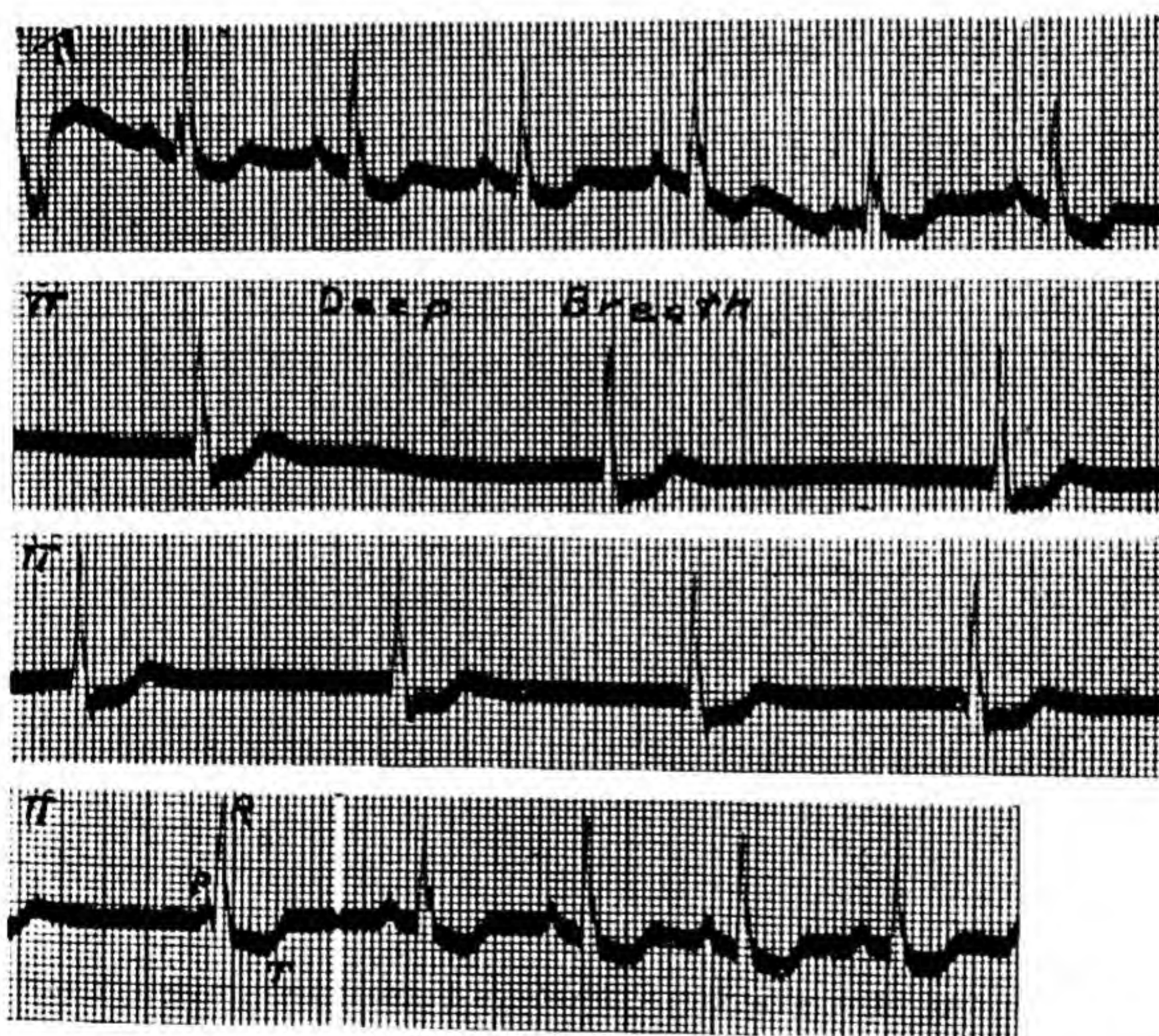


Fig. 11 —Sinus Pauses. This is a continuous tracing from a man with aortic stenosis and angina pectoris. Note that on taking a deep breath the heart slowed markedly, the P waves disappeared, there was an idioventricular rhythm and a gradual return to normal

Similar electrocardiograms and syncopal attacks can occur in some individuals with aortic stenosis or in those who have a sensitive carotid sinus. Figure 11 shows the effect of a deep breath in a man with aortic



stenosis. The whole heart slowed and the patient grew somewhat faint. Figure 12 shows how readily the heart can be slowed by light pressure over the carotid sinus in some individuals. This man was quite well except that he had frequent spells of unconsciousness which came without

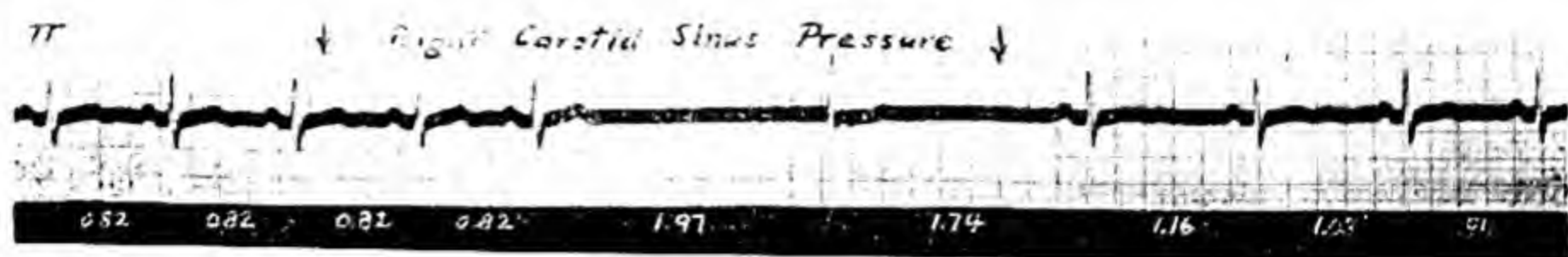


Fig. 12.—Sinus Pauses. From a man, aged sixty, who had syncopal attacks but no heart disease. The right carotid sinus was very sensitive. Note marked slowing of the heart from the carotid sinus reflex.

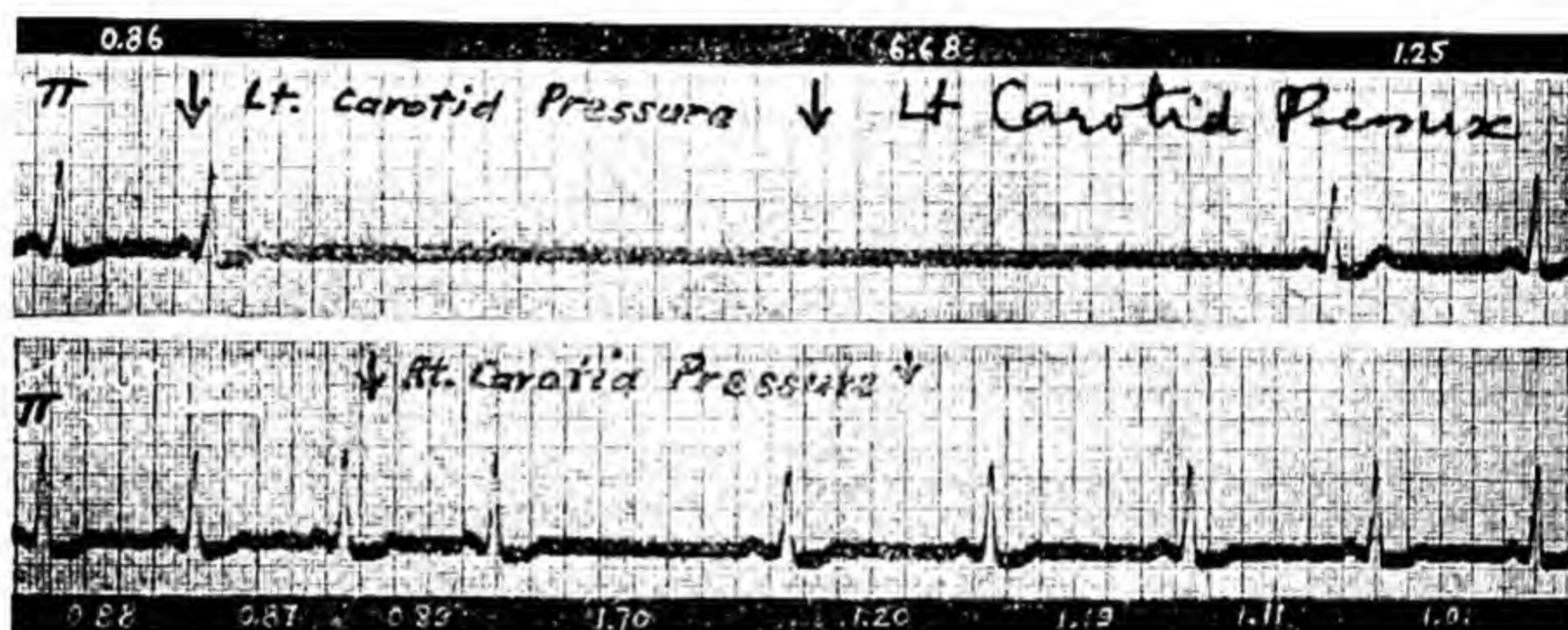


Fig. 13.—Marked Carotid Sinus Sensitivity. Male, sixty years old, with hypertension, aortic stenosis and angina pectoris. Note the prolonged asystole of over six seconds following left carotid pressure. The effect is less marked on the right.

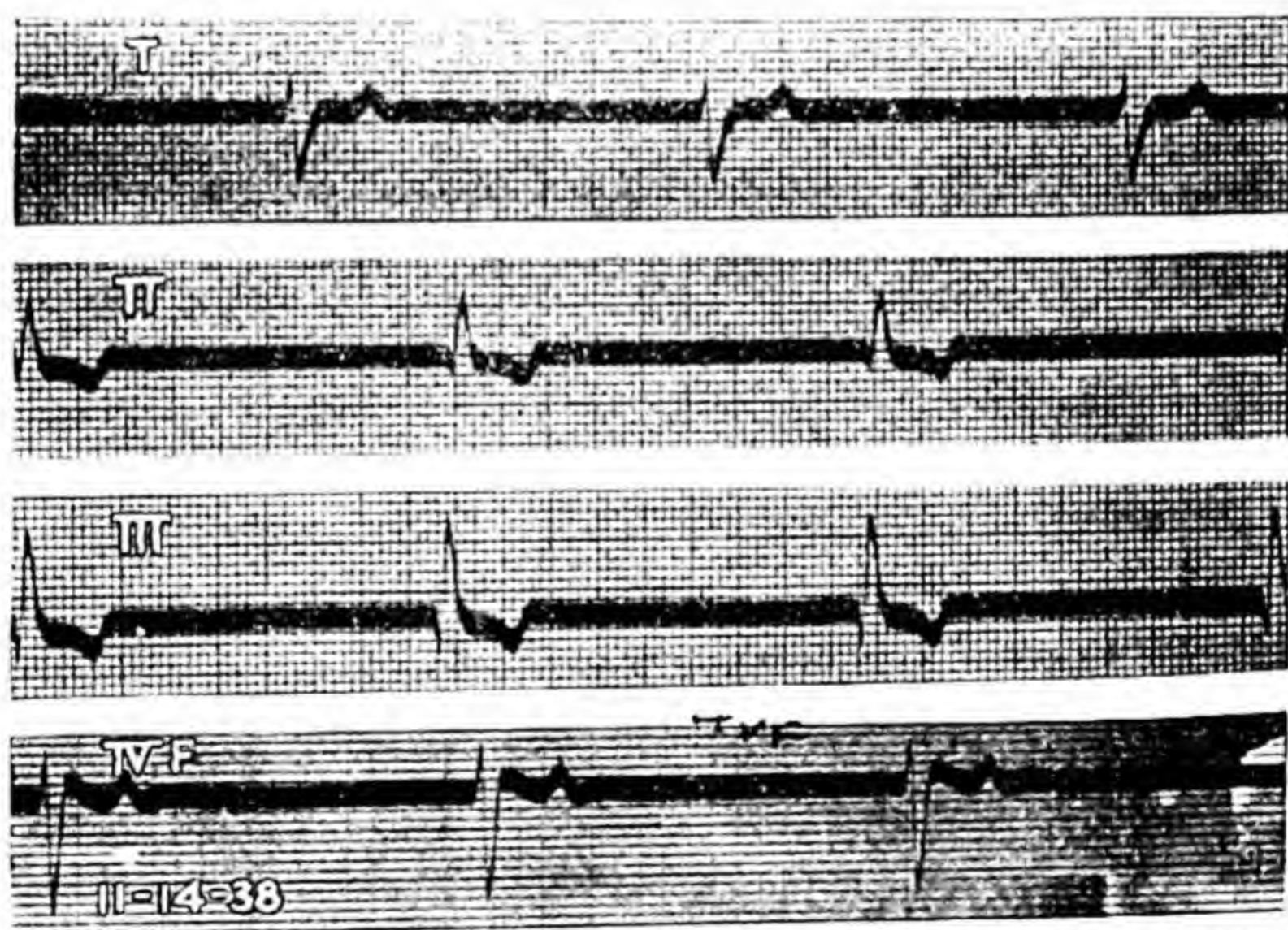


Fig. 14.—Auricular Standstill. Male, seventy years old, who had taken excessive digitalis and developed unconscious spells. Note complete absence of P waves and ventricular rate of 38. Patient made complete recovery with reappearance of normal P waves.

warning. He was entirely cured by taking  $\frac{3}{8}$  grain (0.025 gram) of ephedrine sulfate two or three times a day. Another instance of marked carotid sensitivity is shown in Figure 13.



**Auricular Standstill.**—On rare occasions electrocardiograms are seen in which the auricular complexes entirely disappear for brief or considerable periods of time. This condition is called auricular standstill or inhibition of the auricles (Figs. 14 and 15). The ventricular beat is maintained by the idioventricular pacemaker. Such curves are occasionally observed during quinidine or digitalis administration and indicate a toxic effect of the drug. Syncopal or Adams-Stokes attacks may occur in patients manifesting this disturbance and it is imperative that the drug causing the auricular standstill be omitted.

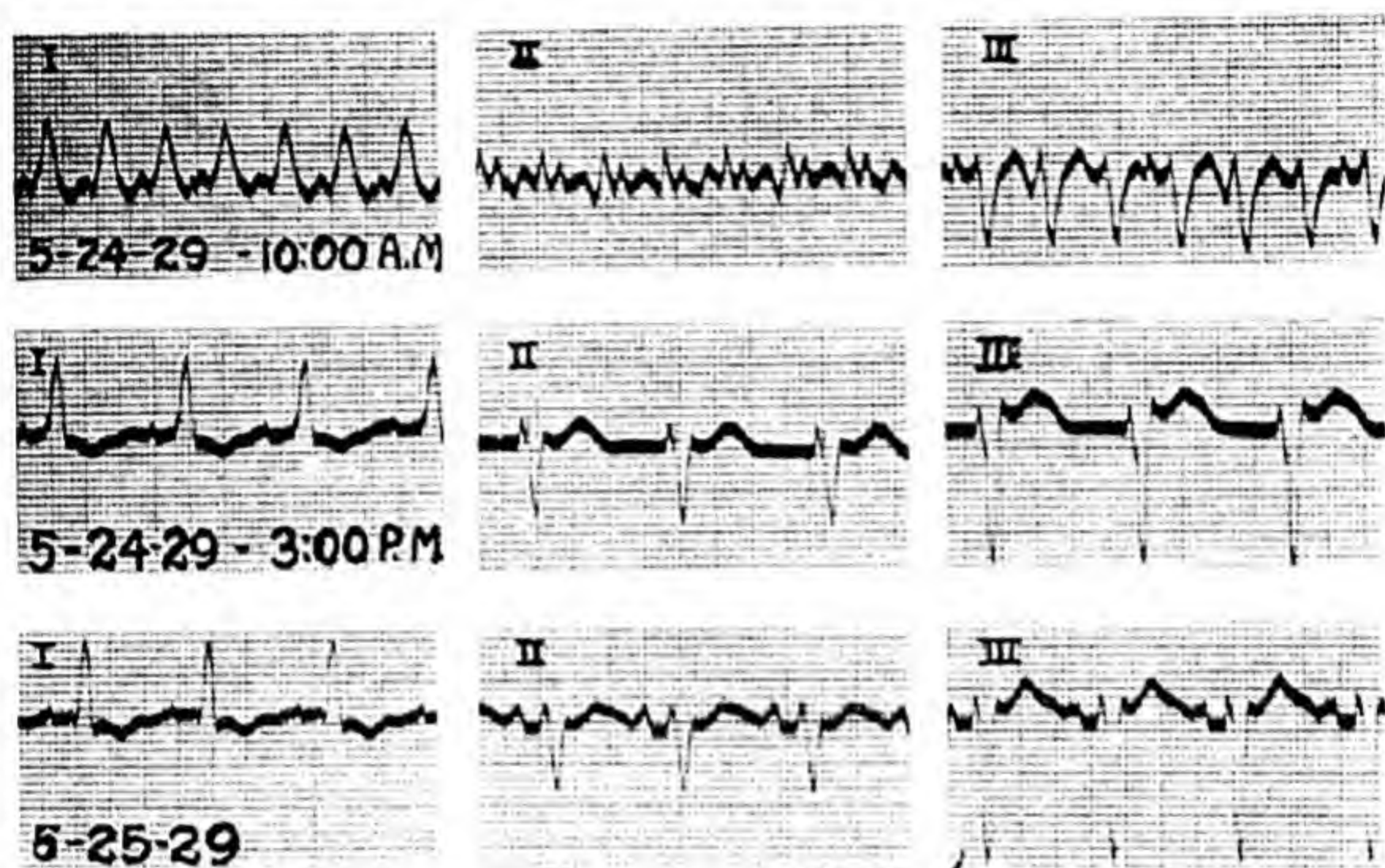


Fig. 15.—Auricular Standstill Due to Quinidine. Upper tracing shows paroxysmal ventricular tachycardia with a ventricular rate of 169. Middle set shows auricular standstill in Leads II and III. Lower set shows return of auricular activity. The patient had received a single dose of 1 gram of quinidine sulfate at 11.30 a. m. on May 24, 1929.

### ECTOPIC RHYTHMS

In the preceding paragraphs we discussed disturbances in the pacemaker of the heart. The second major abnormality in the mechanism of the heart beat is the formation of ectopic rhythms. Impulses can arise in almost any part of the heart: auricles, junctional tissue or ventricles. When such ectopic beats occur they interfere with the normal sequence of events and produce peculiar and characteristic electrocardiographic changes. The fundamental principle underlying these alterations is that if an impulse travels an abnormal course through a certain portion of the heart, the electrocardiographic representation of that impulse will be abnormal.

**Premature Auricular Beats.**—Impulses may arise in any portion of the auricular musculature. Ordinarily the tendency for beats to arise in abnormal parts of the heart is held in abeyance, because the pace set by the sino-auricular node is faster and prevents other foci from functioning. Under abnormal conditions ectopic foci are enabled to initiate impulses and when such isolated beats occur they are called *premature* or *ectopic*



*beats* or *extrasystoles*. On listening over the precordium one hears a regular rhythm (lub-dub, lub-dub) and then suddenly a quick beat followed by a pause, after which the regular sequence is restored. This premature beat may come only at very rare intervals or as frequently as every second or third cycle. Although the beat can be heard over the precordium it may produce such a small pulse wave that it is only barely felt at the wrist or it may be entirely imperceptible. This is so because the heart contracts early in diastole when the volume of blood in the ventricles is quite small.

From an electrocardiographic point of view we must visualize the wave of excitation as arising in some point in the auricle more or less distant from the sino-auricular node. The course through the auricle that

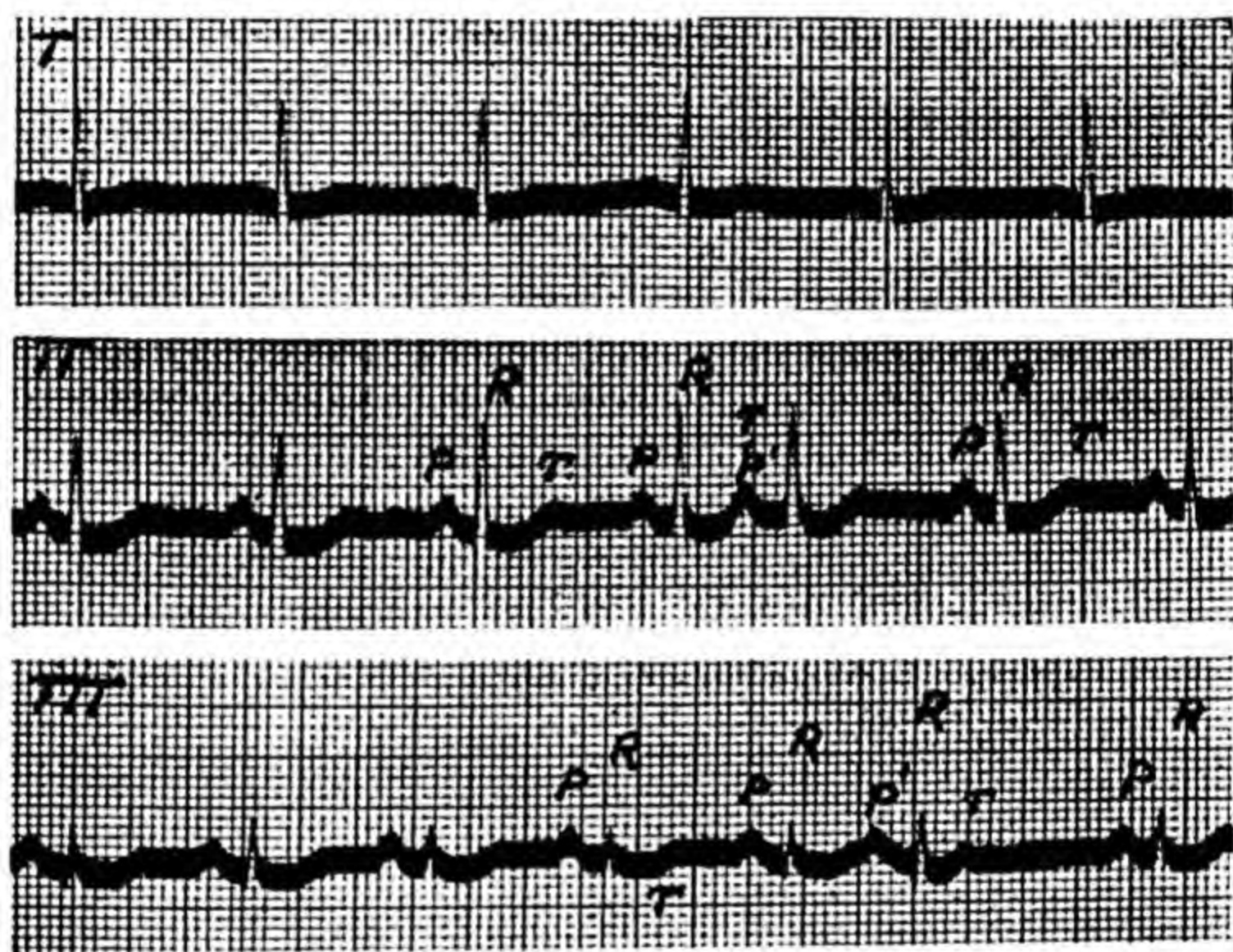


Fig. 16.—Premature Auricular Beats. Note that the irregularity is due to an occasional premature beat ( $P'$ ) which becomes superimposed on the previous T wave. The ventricular complexes following the premature beats are of normal form (Leads II and III) From a man who had a benign irregularity of the heart.

this impulse must take will be abnormal. It will travel possibly from left to right rather than from right to left or upward rather than downward. The P wave which represents auricular activity will have to be abnormal in form in one or more leads. How different from the normal P wave it will be, will depend upon how far away the ectopic focus is from the normal pacemaker. It may, therefore, remain upward or become inverted (Figs. 16, 17, 18). When this impulse reaches the sino-auricular node it destroys whatever impulse-forming material had been built up there and the node starts over again in the production of a normal beat. The premature beat is also traveling downward through the auriculoventricular junctional tissue to reach the ventricles. The pathway of this beat through the ventricles therefore should be normal and the QRS-T complex will be of normal form. Generally this is so (Figs. 16 and 18). At times the ventricular complex following a premature auricular beat, however, is ab-



normal (Fig. 17) and the P-R interval may be delayed. In fact, on rare occasions the beat may be blocked and then it is called *blocked premature auricular beat*. This occurs because the beat has come so quickly that the tissue has not recovered completely and there may be a slight delay in one part or another of the conduction apparatus distorting the spread of the wave of excitation. The premature and abnormal P wave may need to be sought for carefully. It often is hidden on the previous T and is to be detected by a slight alteration in the height or configuration of this T wave (Fig. 16). At other times because the ectopic focus is so near the normal pacemaker the P waves differ only very slightly from the normal ones (Fig. 51, lower curves). Occasionally a premature auricular beat

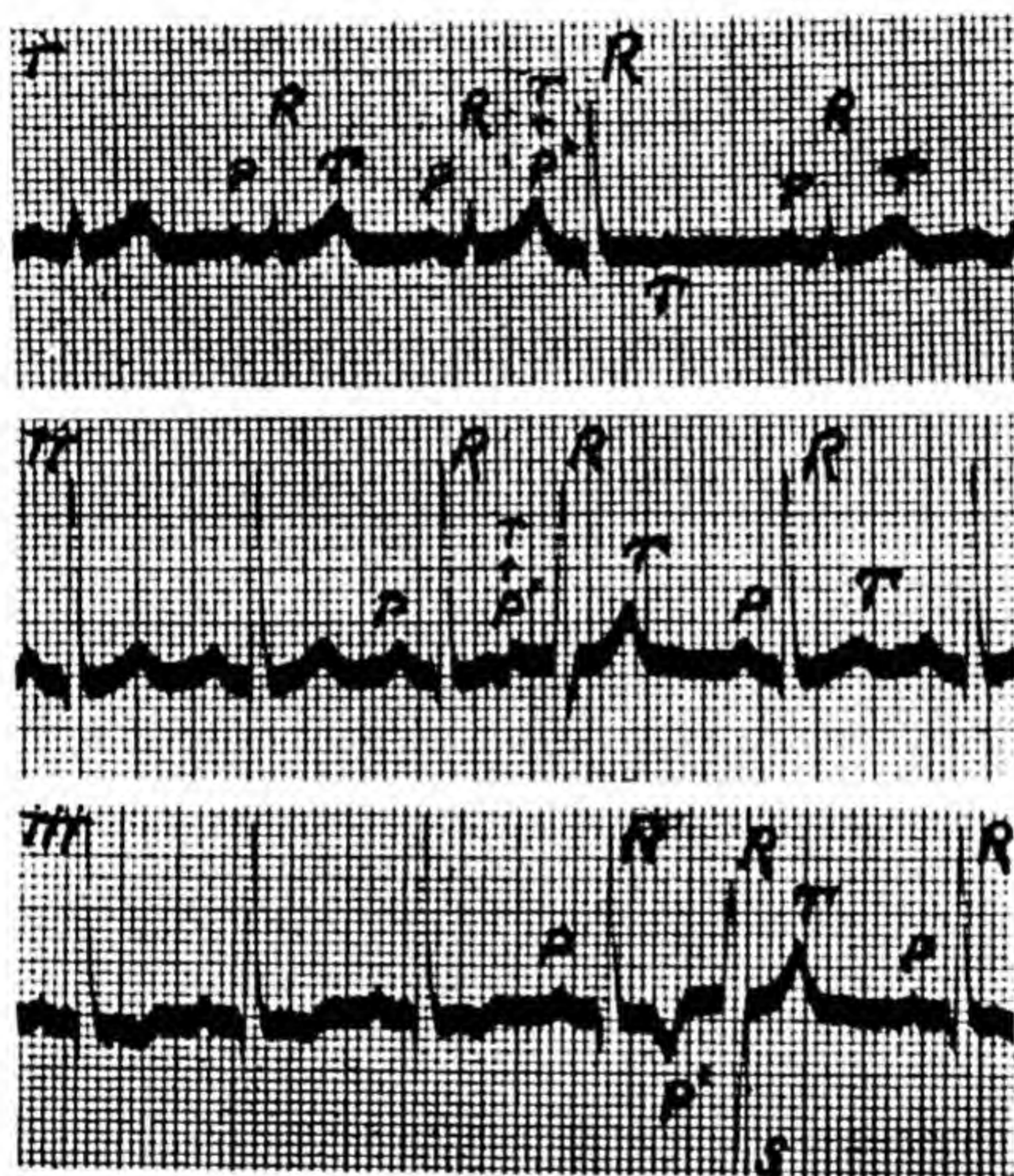


Fig. 17.—Premature Auricular Beats. From a young man with no heart disease. Note that the premature  $P'$  waves fall on the preceding T waves are inverted in Leads II and III, and are followed by ventricular complexes of abnormal form.

produces no electrical disturbance in one of the leads but the finding of a P wave of an abnormal form in one of the other leads will reveal its abnormal origin.

Premature auricular beats can readily be distinguished from those of ventricular origin. Even when the former are followed by abnormal ventricular complexes, the QRS waves are not as broad as in the latter condition. The detection of the preceding abnormal P wave identifies the beat as auricular. Finally, the pause after an auricular extrasystole, although longer than the normal cycle, is generally not completely compensatory, *i.e.*, the length of the two cycles including the extrasystole is less than two normal heart cycles, whereas with ventricular extrasystoles



it is equal to two normal beats. Occasionally, however, even premature auricular beats are followed by completely compensatory pauses.

Premature auricular beats are fairly common and do not by themselves indicate heart disease. They occur in individuals otherwise well as a

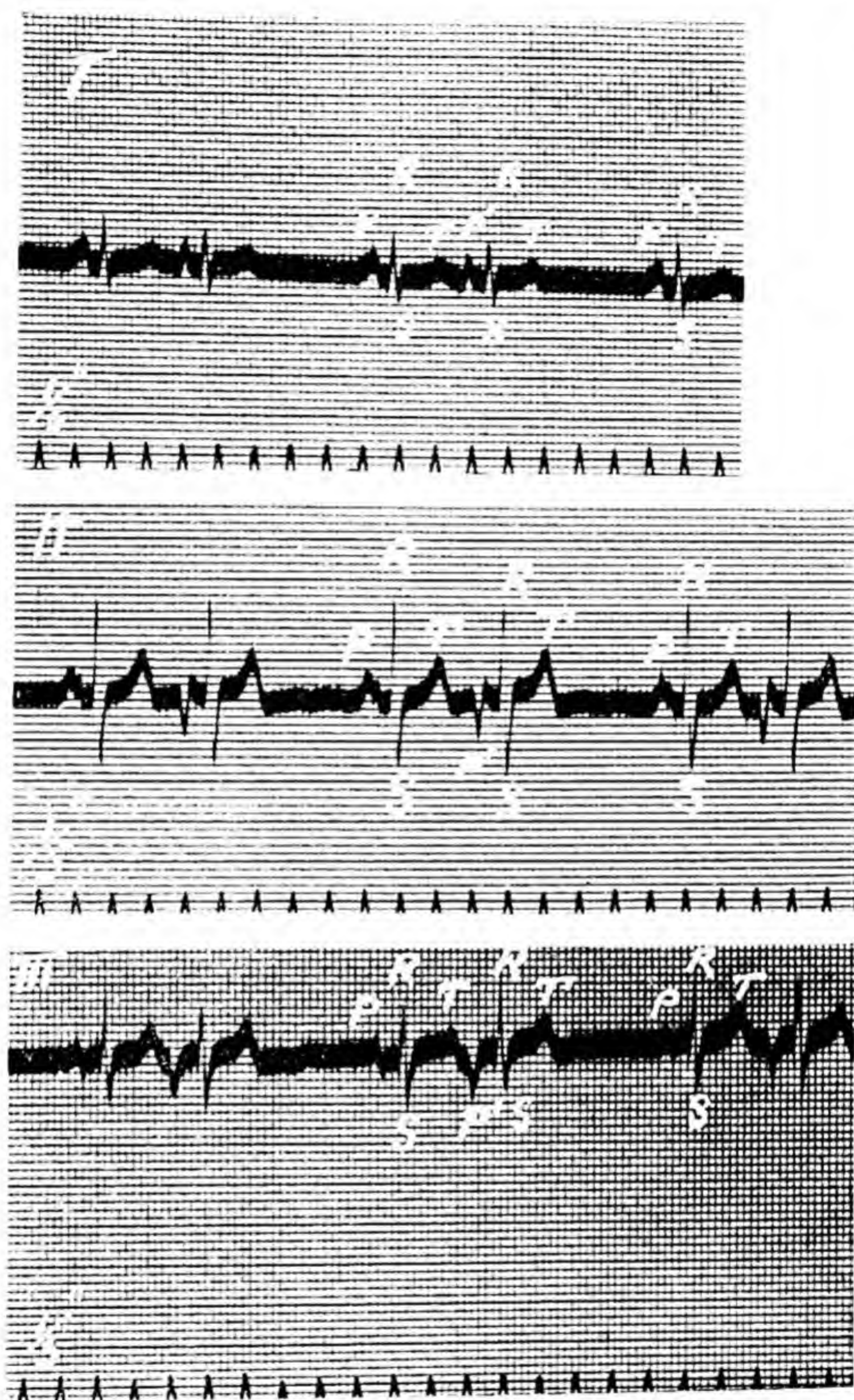


Fig. 18.—Premature Auricular Beats. Note that every second beat is a premature  $P^1$  wave which is upright in Lead I but inverted in Leads II and III. This is a form of coupled rhythm. (Author's article in *Oxford Loose-Leaf Medicine*, vol. II. Courtesy of Oxford University Press.)

purely functional or neurogenic disturbance and need produce no symptoms or disability. When such is the case, the irregularity should not be treated, nor should the patient be restricted in his activities. When these beats produce a good deal of palpitation and are annoying, quinidine or digitalis may be helpful. Occasionally potassium salts (potassium phos-



phate or chloride 2 grams three times a day by mouth) may prove beneficial. Unlike ventricular extrasystoles premature auricular beats do not result from excessive digitalis. They do occur in patients with organic heart disease, but the diagnosis of structural disease will then have to rest on other evidence. There is some association between this type of extrasystole and auricular disturbances of higher grade, *e.g.*, tachycardia, flutter and fibrillation. Patients who show the latter irregularities often are found to have premature auricular beats at other times. On following the progress of a case of mitral stenosis this form of extrasystole often occurs for some years preceding the development of auricular fibrillation, and so the detection of the former may lead one to suspect that the latter irregularity will not be long delayed. Likewise, if a patient has paroxysms of some form of rapid heart action, the finding of an occasional auricular extrasystole between attacks helps to throw light on the nature of the paroxysms that have occurred in the past.

**Paroxysmal Auricular Tachycardia.**—As one continues the consideration of ectopic rhythms in the auricle, a disturbance of somewhat higher degree is paroxysmal auricular tachycardia (Figs. 19 to 28). Here, in-



Fig. 19.—Paroxysmal Auricular Tachycardia. Note the complete cycle of a brief attack of tachycardia, lasting five seconds.  $P^1$  is different in form from the normal P wave, but the ventricular complexes are unchanged. The onset and offset are instantaneous, the rhythm is perfectly regular, rate is 158. The patient was a girl of twenty-one without heart disease who complained of palpitation.

stead of an occasional impulse arising in some abnormal part of the auricular wall, this focus sends out a series of regular impulses. The pace is then set by this ectopic focus and it may be maintained for variable lengths of time, persisting for hours or days and rarely for weeks. A very new concept concerning the nature of paroxysmal auricular tachycardia is developing at present. It is now thought by some observers that a circus movement which has its pathway through the a-v or s-a node is responsible for this abnormality. Inasmuch as the origin of the impulse is abnormal the course it takes through the auricles is abnormal and in so far as an auricular complex can be identified it will differ from the normal P wave. The course of this wave through the ventricles, however, is normal and therefore the QRS waves will be the same as when the heart is beating normally. Figure 19 represents a complete brief paroxysm of this type lasting only four to five seconds. It shows that the onset and offset are abrupt and the auricular waves change their form when the paroxysm starts, but that the ventricular complexes remain the same. It is characteristic of this condition that the rhythm is perfectly regular, much more so than in a normally beating heart. It is also pecul-



lar that in most cases the P waves cannot be easily identified, for they probably are fused with the preceding ventricular complex. The result

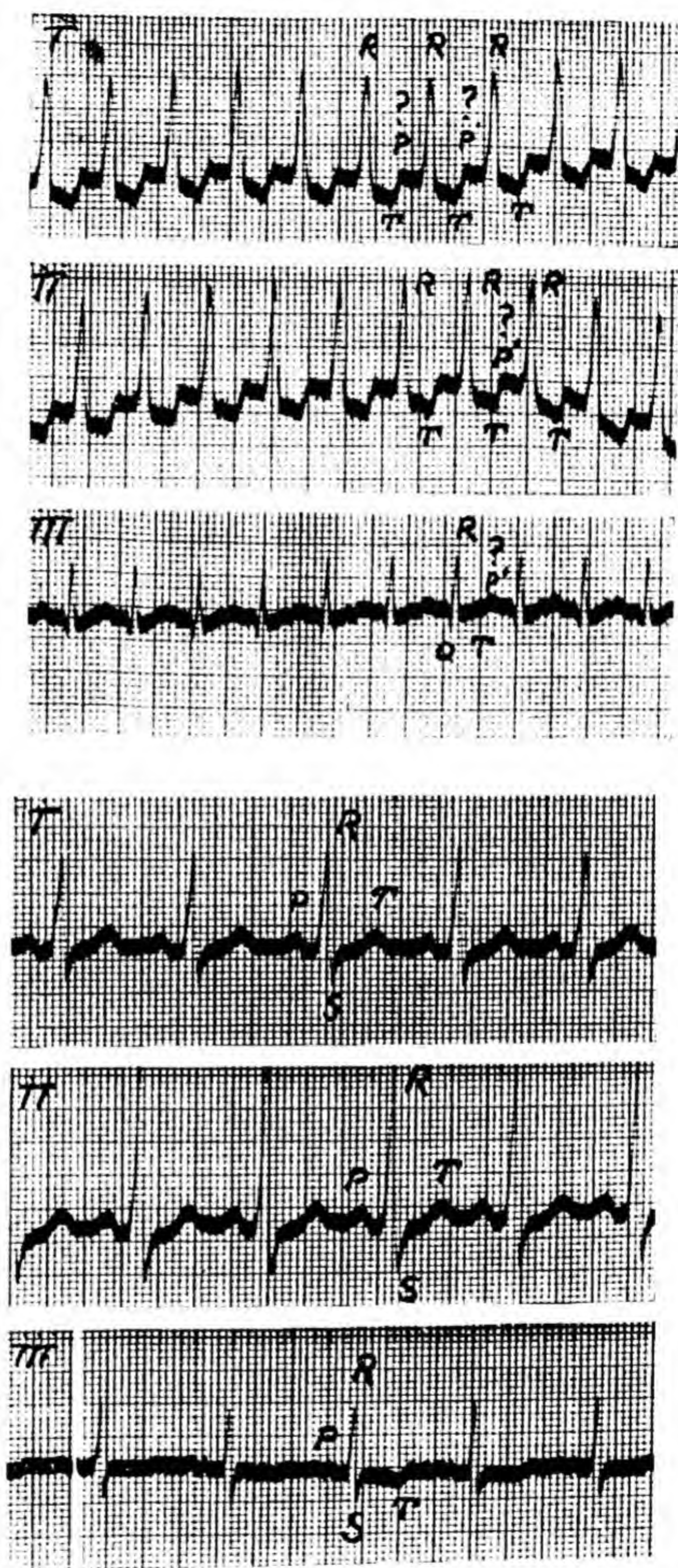


Fig. 20.—Paroxysmal Auricular Tachycardia. Upper three leads show a perfectly regular rate of 172. Auricular complexes ( $P_1$ ) probably buried in the previous T waves. Lower curves on the same patient taken after attack was over show a normal slow rhythm, rate of 83.

is that one often sees only what appears to be an R and T wave coming in rapid succession.



Clinically this condition is important as it is common, can cause a good deal of discomfort and concern and on rare occasions can have serious consequences. It is also important from a practical point of view because it can readily be recognized at the bedside and effectively treated. It occurs for the most part in an individual who has a structurally normal heart. Occasionally it is a complication of organic heart disease and then may produce alarming symptoms. Ordinarily attacks occur without any detectable cause. The patient is apt to blame some particular happening



Fig. 21.—Paroxysmal Auricular Tachycardia. (Same case as Fig. 20.) Note the abrupt cessation of the attack by pressure over the left carotid sinus. The irregularity of the base line is due to movements of the patient.  $V_x$  indicates ventricular extrasystoles which are common during the transitions. Patient had hypertension and angina pectoris.

that took place just before the attack, such as a motor ride or eating a certain kind of food. Indigestion or gas is often suspected as being the cause, and yet treatment directed at the gastro-intestinal tract or the diet will be of no avail. A careful history will frequently bring to light the fact that a certain specific event may act as a trigger in setting off a paroxysm. A sudden turn of the head, or bending over to tie one's shoes, an unexpected emotional upset or a dream all have been known to precipitate such spells. On the other hand many attacks occur without any obvious cause.



Fig. 22.—Paroxysmal Auricular Tachycardia. Cessation of attack by right carotid sinus pressure. Note long pause followed by resumption of normal rhythm. Rate of attack 158. P waves during attack not easily identified. Patient had no heart disease.

The attack itself is instantaneous in onset and offset (Fig. 19). In most cases the patient is aware of this and will describe it as coming suddenly and stopping with a "thump." At times, although the changes are sudden, the sensations will be described as occurring quickly but not abruptly. The patient will complain of palpitation or fluttering of the heart, become uneasy, nervous and apprehensive and want to lie down. Occasionally there is pain over the heart and there may be typical anginal distress even with radiation to the arms. Sometimes nausea and vomiting occur and with it attacks often end. This sort of experience







to detect very minor alterations in rhythm. Not only is the heart rapid and regular but its rate is very fixed for long intervals of time and cannot be altered by simple procedures like breathing or exercise which affect the rate of the normal heart. Over the course of hours the rate may change but not during short intervals of time. The bedside application of this peculiarity is to count the rate carefully for sixty seconds by auscultation over the precordium. This should be repeated in several minutes, preferably under different circumstances, after slight exercise or with the patient sitting up rather than while lying down. If there is a significant difference in the two rates, one can be fairly sure it is not paroxysmal auricular tachycardia. With the latter the first count might be 169 and the second 168. If the second count were 174, it would point to a normal sinus tachycardia (Fig. 6). The difference should be no greater than two beats if the count is made carefully. Even this difference is to be accounted for by the difficulty in timing the first and last beats as one follows the second hand of the watch in its complete circuit. One often hears the statement that the rate, though regular, was too rapid to be counted. Although this may be so when the rhythm is irregular, I believe it means careless observation or lack of interest in accurate counting when it refers to a rapid regular rate. I had the opportunity of testing this in a case that showed a heart rate of 250. Three independent observers counted rates of 248, 249 and 250. When the rate is over 200 the actual counting process may be facilitated by tapping on the table with a pencil synchronously with the heart beat and then counting the taps. Emphasis is placed on this because determining the constancy of the rate can be carried out by any physician and helps to distinguish a normal from an abnormal tachycardia.

The various methods that are used to stop an attack also serve as diagnostic procedures, for there is no other type of rapid heart action that can be made to return to a normal rate by such simple means and so quickly. Not only does a normal tachycardia reach its rapid rate gradually, starting from about 70 or 80 and then increasing to 100, 120 and finally 160 or 170, but when it returns to normal it does so gradually. If vagus stimulation is produced by pressure over the carotid sinus or the eyeball during paroxysmal tachycardia, the rate will remain entirely unaltered or it will abruptly fall to normal, *i.e.*, the rate may change from 190 to 80 in one or two beats (Figs. 21, 22, 23, 35). With normal tachycardia, there is apt to be temporary slowing with gradual return to the previous rapid level (Figs. 35, 70). A similar effect is generally obtained if the tachycardia is due to auricular flutter (Fig. 30) and when ventricular tachycardia is the cause of the acceleration vagus stimulation produces no effect whatever. It is evident, therefore, that the diagnosis can generally be made at the bedside and only rarely will electrocardiograms be necessary.

The *treatment* of the condition can be divided into two problems. The first and easier task is to stop the attack. The second and more difficult



is to prevent its recurrence. Most attacks will end if left alone and no harm will result. Occasionally severe congestive heart failure or anginal pain may develop and rarely may prove to be fatal. For thirty years I have followed one extraordinary patient who had yearly severe attacks in 1911, 1912 and 1913. During one of these he developed aphasia, which cleared in several months, during another he had a hemiplegia which disappeared in a few months leaving only a very slight spasticity, and during the third, dry gangrene requiring amputation of the left arm at the shoulder. The tachycardia in these spells lasted from five to eleven days incessantly. When I saw the patient during his fourth attack the heart rate was 250. The blood pressure was about 94 mm. systolic and 88 mm. diastolic. He developed a fever of  $101^{\circ}$  and an appreciable leukocytosis. He also complained of a constant distressing constricting pain in the chest. There were a few râles at the bases of the lungs. Many of these features resemble those of an acute coronary thrombosis, a condition with which any severe form of paroxysmal rapid heart action can be confused. In fact, the heart was found to have dilated 2.5 centimeters as compared to its size on x-ray examination after the attack was over. Such dilatation is unusual in paroxysmal tachycardia, for in most cases there is no dilatation or very little. It is quite evident that proper treatment was very urgent in the case just cited. At first it was found that carotid sinus pressure was ineffective but vigorous pressure over the eyeball ended the attack immediately. Subsequent attacks were readily controlled by pressure over the carotid sinus and the patient was taught how to perform this procedure. For some unknown reason the attacks gradually became quite rare. The patient remained in excellent health for over twenty years but lately has had typical mild anginal pain on effort which is not associated with attacks of tachycardia.

Further illustrations of the urgency of proper treatment are the rare instances in which attacks occur in patients on the operating table during anesthesia. Breathing may cease entirely and the patient may become pulseless. Even fatalities may occur. I have seen several cases in which the condition seemed critical but was readily controlled by simple methods.

The simplest means of stopping an attack is to have the patient hold a deep inspiration as long as he can. He might be advised to make a quick expiratory effort with the glottis closed after a deep breath is taken. Many attacks can be ended quite readily in this way. The patient should be told that if this fails, he might try to induce vomiting by placing his finger down the pharynx. The act of gagging, retching or vomiting often ends attacks. This may be brought about by the hypodermic injection of apomorphine. More effective still are 1 to 4 teaspoonfuls of syrup of ipecac. Some have learned that lying down or lowering the head may be effective. Often when the attack continues, morphia is given and during the subsequent hour or two the heart returns to normal. When this occurs it is difficult to say that the medication causes the attack to end. The most valuable treatment is carotid sinus pressure. This is per-



formed by placing two fingers over the carotid artery just below the jaw, where a slight bulge is felt as the common carotid artery divides, and pressing and massaging the artery against the spine for several seconds. First one side and then the other may be tried but not both at the same time. This stimulates the vagus nerve reflexly (carotid-sinus reflex) although it used to be thought that the vagus nerve which lies just behind the artery in the carotid sheath was stimulated directly. In the majority of cases, when this is properly done, the attack will end instantly. If this fails, ocular pressure may be tried (Fig. 23). One eye at a time should be firmly pressed backward. To be effective the pressure

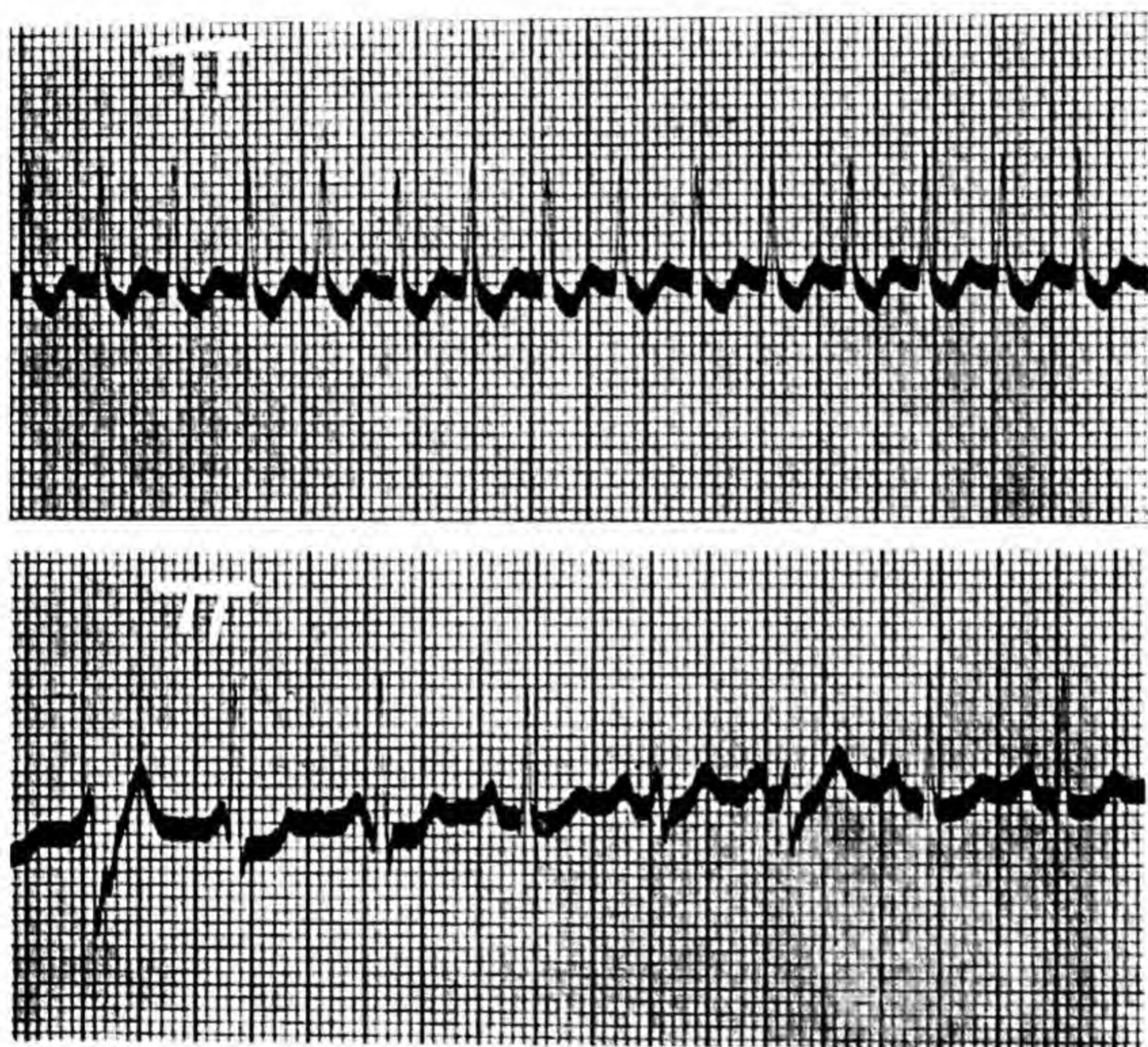


Fig. 25.—Paroxysmal Auricular Tachycardia Arrested by Mecholyl. Upper tracing shows regular rate of about 225. Lower set taken one minute after 25 milligrams of mecholyl was injected subcutaneously shows slow rate with occasional extrasystoles. The patient was a woman thirty-six years old with no organic heart disease. Note electrical alternation of the R waves during tachycardia.

will have to be painful, but this method may succeed where others have failed. It is particularly applicable if the patient is already under an anesthetic for then the pain will not be felt. In this procedure the reflex goes up the fifth cranial nerve and down the vagus (oculocardiac reflex). There have been rare instances in which carotid sinus pressure has resulted in hemiplegia. I have seen one such case in an elderly man. Very likely when marked cerebral sclerosis is present the fall in blood pressure and cessation of the circulation may result in local cerebral thrombosis. In fact, in extremely rare cases, fatalities have occurred because after a pause in the heart the normal beat was not resumed.



Finally, there are drugs that may stop attacks of tachycardia. Recently acetyl- $\beta$ -methylcholine or mecholyl has been found to be very effective (Fig. 25). The average adult dose is about 20 milligrams, given subcutaneously. This produces a rather powerful stimulating effect on the vagus apparatus. It often aborts an attack in a few minutes. Because of occasional untoward reactions, atropine should always be available for immediate use in doses of 1 to 2 milligrams given hypodermically. Asthmatics or strongly allergic individuals should not be given mecholyl. Another drug that I have employed in former years when all other methods failed and the condition of the patient seemed critical is quinidine sulfate administered intravenously (Fig. 24). The dose that was used was 0.3 gram (5 grains). There is some danger of instant fatality from the

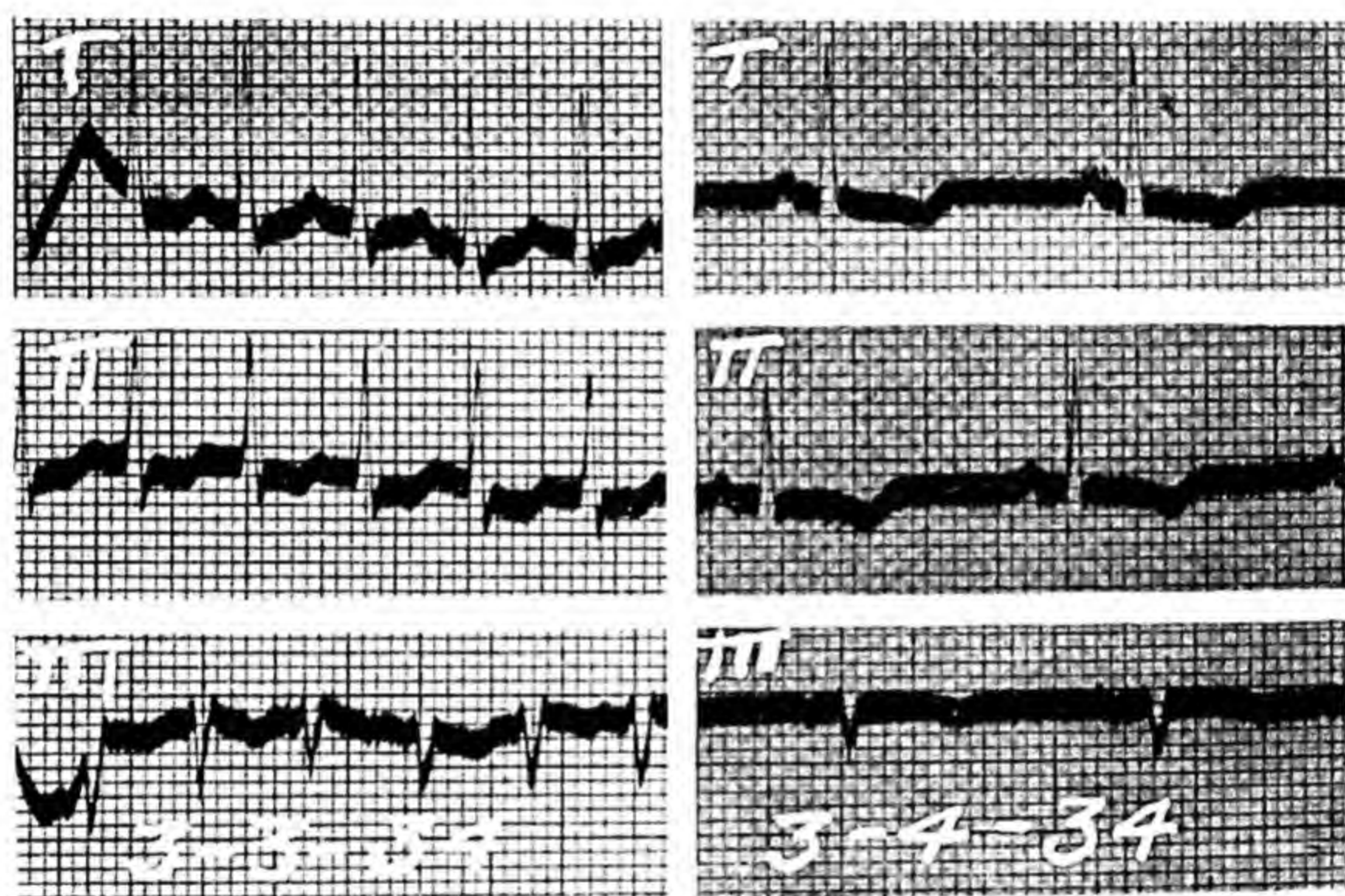


Fig. 26.—Paroxysmal Auricular Tachycardia Stopped by Digitalis. Tracings on the left show regular rate of 196. Set on the right taken twenty hours later, one gram of digitalis having been given orally, shows normal rate of 69. The patient was a man sixty-two years old with bronchopneumonia and a fever of  $103^{\circ}$  F., who recovered satisfactorily.

intravenous use of quinidine, so it cannot be recommended unless the circumstances warrant the risk. Until recently quinidine preparations have not been available for parenteral use. The following procedure, however, could be employed. A mixture is made of 0.3 to 0.5 grams of quinidine sulfate in 20 to 30 cc. of water. To this a few drops of concentrated hydrochloric acid must be added to keep it in solution. After boiling, the solution may be slowly injected intravenously. It has also been suggested that the required amount of quinidine may be dissolved in 200 to 300 cc. of saline. This slower method of injection may be safer. Ampules containing 0.6 gram of quinidine hydrochloride have now been prepared and are suitable for intramuscular or intravenous use. Occasionally large doses of digitalis (0.5 to 1.0 gram given parenterally or orally) may gradually arrest an attack (Fig. 26). Most recently magnesium sulfate in doses of about 2.0 grams given intravenously has also



proved successful (Fig. 27). There is a slight risk with this method as there is with all intravenous medication for tachycardia. In general it will be found that the paroxysm can be controlled by one method or another.

The prevention of attacks presents a more difficult problem. If they recur infrequently, once a year or so, it hardly seems wise to institute a course of drug therapy because, not knowing when the attack is due, it will be necessary to keep the patient on the medication all those intervening months with the hope of inhibiting this rare spell. It is better to do nothing and then to stop the attack when it occurs. When attacks come frequently the problem is different for they may be very annoying and it is possible to ascertain whether or not drug therapy is effective.



Fig. 27.—Paroxysmal Auricular Tachycardia Stopped by Magnesium Sulfate. Upper tracings show regular rate of 257. Lower set was taken two minutes after the intravenous injection of 3 grams of magnesium sulfate and shows a normal rhythm. The patient was a man sixty-eight years old with probable spinal cord tumor but no heart disease.

Occasionally the constant administration of 0.2 to 0.3 gram (3 to 5 grains) of quinidine sulfate will prevent attacks. Even more effective is constant complete digitalization. This must be carried out just as it is done in congestive heart failure. One or the other of these methods frequently obviates or at least diminishes the frequency or severity of the attacks.

Although, in most cases, heart block is not associated with paroxysmal auricular tachycardia there is a small group in which auriculoventricular block of some degree does exist (Fig. 28). This type differs from the more usual one in that it is less responsive to the measures that ordinarily stop attacks, it may persist for long periods of time, even months or years, and it presents some features that resemble auricular



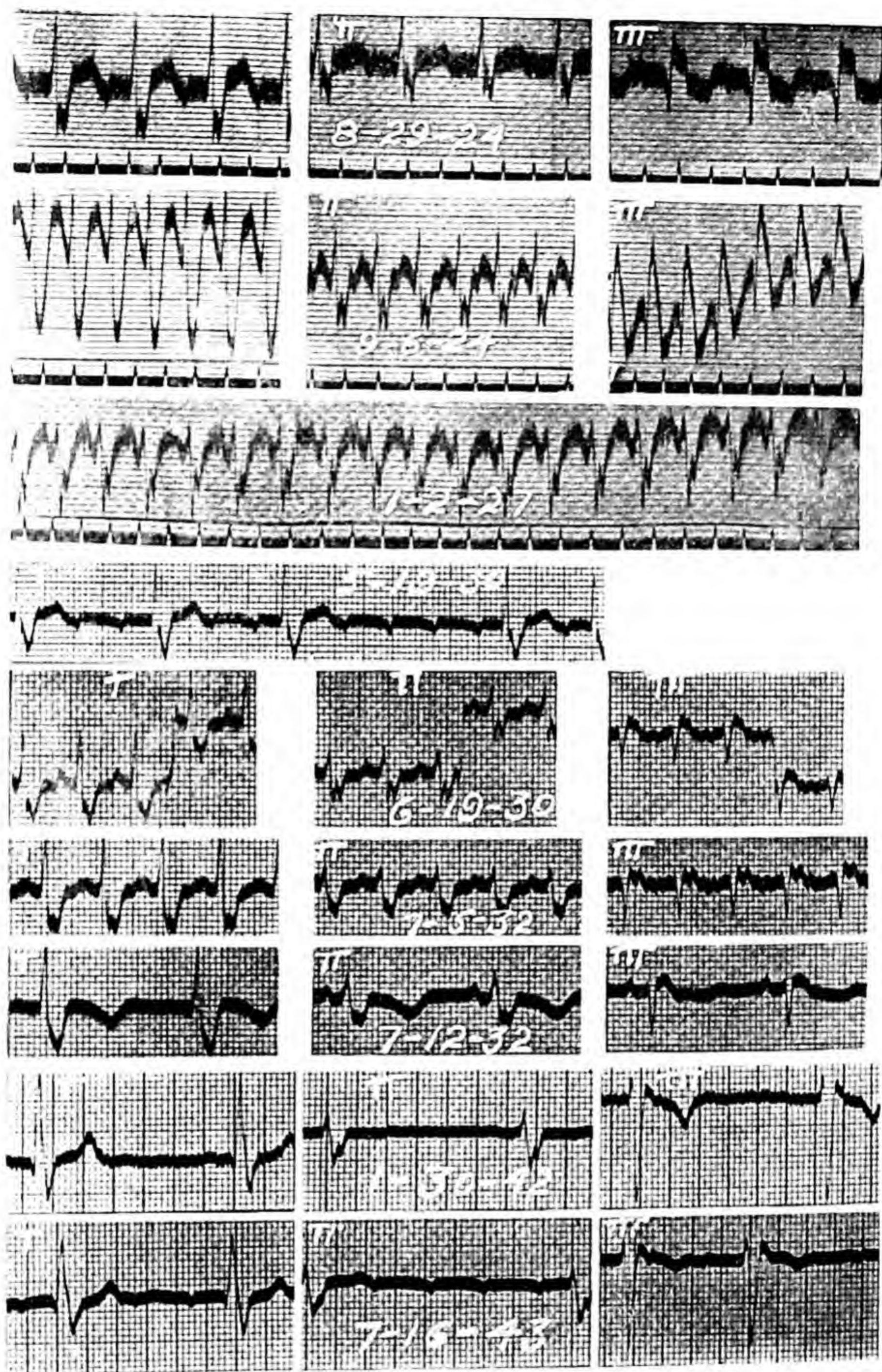


Fig. 28.—Paroxysmal Auricular Tachycardia with Block (Unusual Type). Series of tracings taken over a course of nineteen years. Some show a 1:1 rhythm with a ventricular rate of about 240 (second set). Others show a 2:1 rhythm (first set) with ventricular rate about 120; the fourth and the last sets show varying block with auricles still rapid, regular at about 200, and ventricles slow at about 50; interspersed are sets with normal slow rhythm (seventh and eighth). Right bundle branch block is present throughout. The patient is now seventy-two years old and has been ambulatory without congestive failure all this time.

flutter. In fact, recent studies of this and other types of paroxysmal auricular tachycardia lend support to the idea that some of the cases are due to a circus movement in which the pathway runs through the a-v



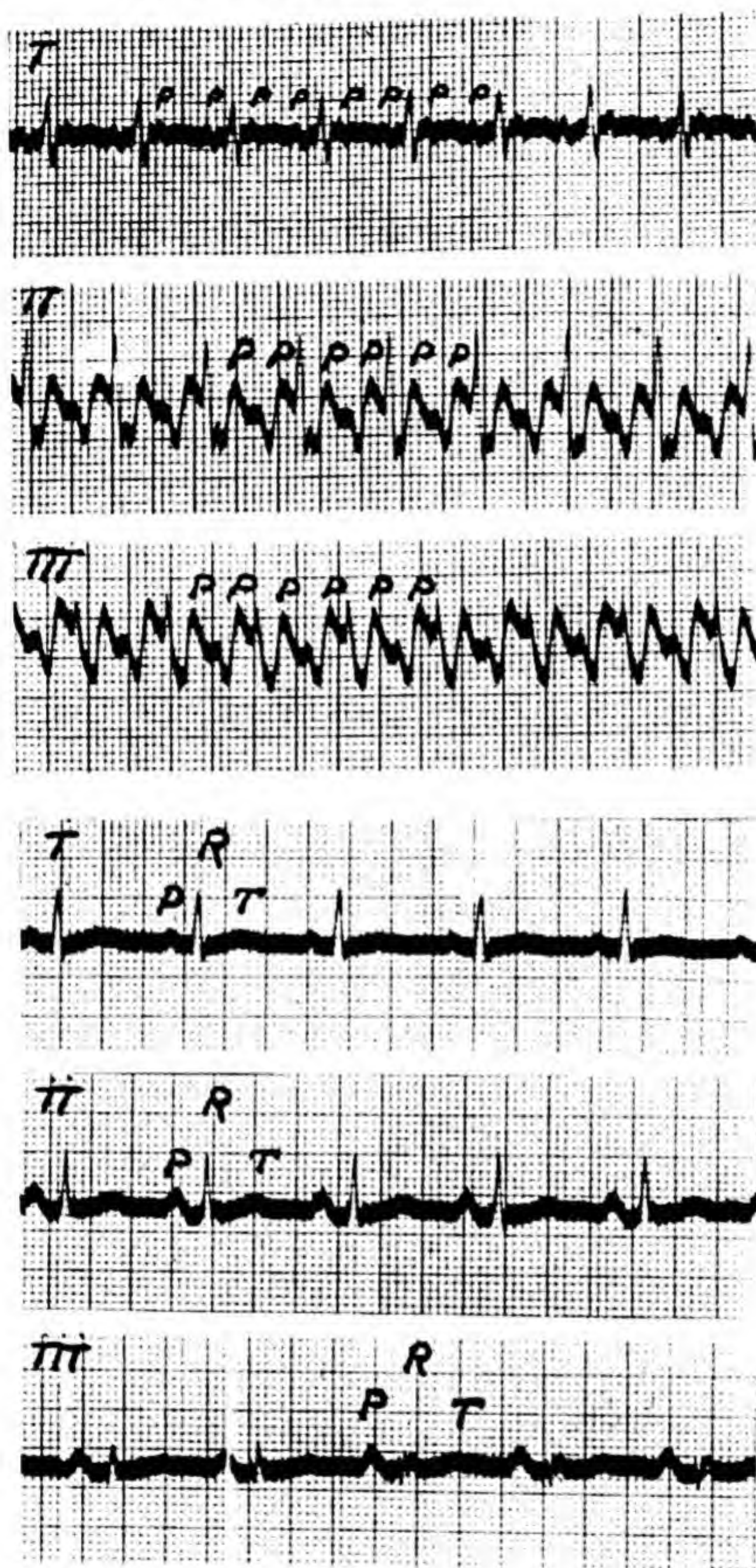


Fig. 29.—Auricular Flutter. Upper three leads taken during auricular flutter, lower tracings taken four weeks later during normal rhythm. During tachycardia the auricular rate is 278, ventricular 139 (2:1 block). Identity and rapidity of auricular beats are difficult to make out until the ventricles are slowed (see Fig. 30). Note the small P waves in Lead I, prominent triangular P waves in Leads II and III with a sharp upstroke and a coarsely notched downstroke. Normal mechanism was established on quinidine therapy. The patient had no heart disease but complained of syncopal attacks for three years.

node. It will be found that quinidine and digitalis are only occasionally effective in controlling this rarer form of tachycardia, although digitalis may at least slow the ventricular rate.

**Auricular Flutter.**—A still higher degree of auricular disturbance is



auricular flutter. In this condition the auricular rate generally is between 200 and 350 and except in very rare instances the ventricles respond to only a fraction of this number, as the junctional tissue cannot conduct impulses so rapidly, *i.e.*, there is in a sense a certain degree of a-v block. In exceptional cases all auricular beats come through to the

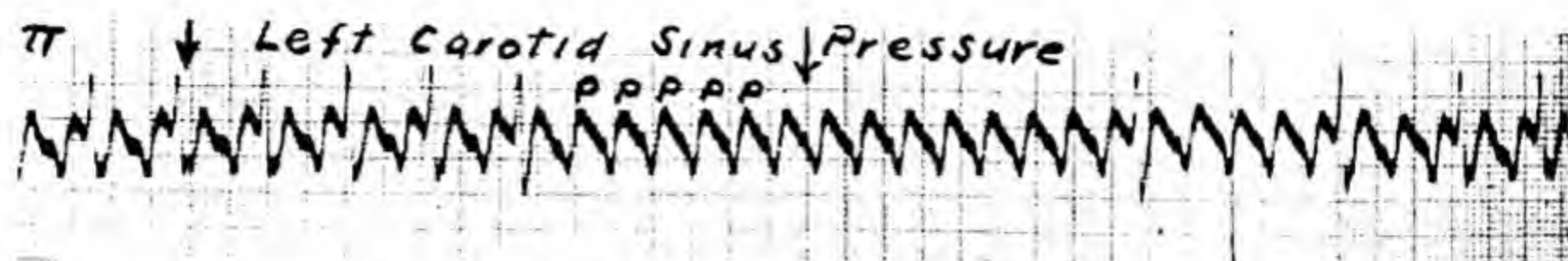


Fig. 30.—Auricular Flutter. Effect of left carotid sinus pressure. (Same patient as Fig. 29.) The ventricular rate is temporarily inhibited, permitting the undisturbed rapid auricular mechanism to become clear.

ventricles and the heart rate is then very rapid (Fig. 33). During quinine therapy the auricular rate may fall to as low as 150. It has been shown that the mechanism of this peculiar abnormality is the development of a circus movement. By this is meant that an impulse gets started and encircles the auricles around the venae cavae and on its re-

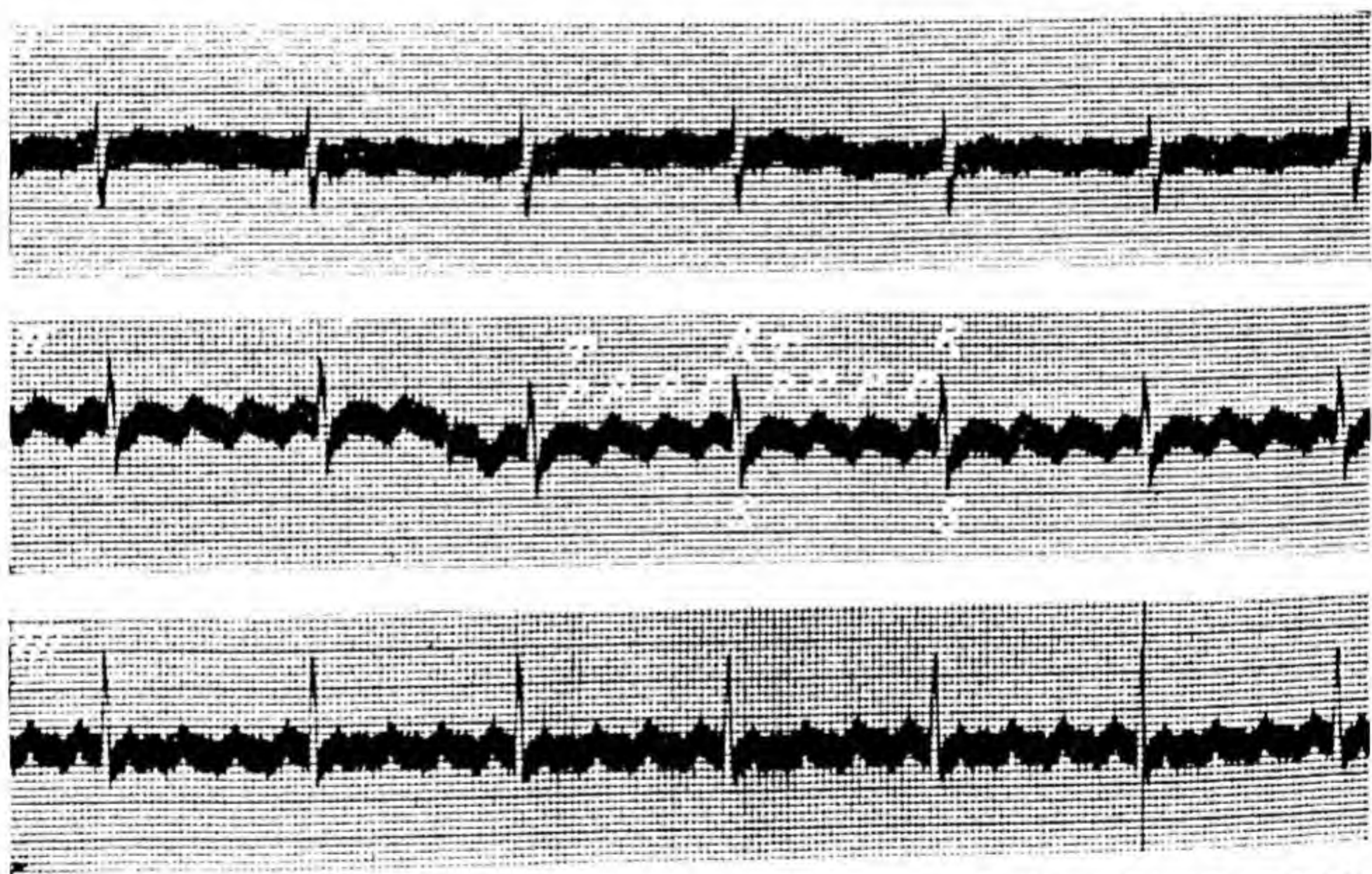


Fig. 31.—Auricular Flutter. The P waves are regular, rate 208; the ventricular complexes are regular, rate 52; there is a 4:1 block. Note triangular form of P waves in Leads II and III. The slow ventricular rate resulted from digitalis. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

turn to the original focus finds the tissue out of its refractory state and continues around again and again, always meeting muscle bundles ahead of it that are ready to conduct the impulse. In auricular flutter this pathway is constant and therefore the waves will be regular and constant in form. In auricular fibrillation, to be discussed later, because the rate is more rapid, each impulse finds bits of tissue ahead of it that are still



refractory following the previous contraction, so that it has to deviate to one side or another constantly seeking fibers that will conduct. The result is a sinuous course and more irregular and more inconstant fibrillary waves occur. Auricular flutter is therefore regarded as a pure circus motion while auricular fibrillation is an impure one.

The electrocardiograms of auricular flutter are very peculiar and characteristic. In Lead I the auricular waves are represented by small notches (Fig. 29). In Leads II and III the waves have a triangular form. The upstroke is sharp and smooth and the downstroke more prolonged and notched at its midpoint. It seems as if there is a hesitation on the downstroke where the iso-electric line might have occurred. It makes one think that each auricular cycle begins at this point and that the entire complex is diphasic rather than beginning either at the top or bottom of these P waves. The flutter waves are continuous; as one cycle ends



Fig. 32.—Auricular Flutter. The ventricular beats (R) come irregularly but because of responses to regularly beating auricles (P) there is equal grouping of beats, *i.e.*, not an absolute irregularity. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

the next begins. There is also a similarity between the flutter waves in different patients. In untreated patients there will generally be found a 2:1 heart block, every second beat failing to reach the ventricles. The auricular rate may be 320 and the ventricular 160. It therefore may be difficult to distinguish the auricular waves or count their rate because they become superimposed on the ventricular complexes. With experience one learns to detect flutter from the appearance of the electrocardiograms but if there is any doubt as to the underlying mechanism, vagal stimulation can help to identify the condition. In Figure 29 it may be questioned whether there are one or two auricular complexes between each ventricular beat. But when the ventricles are inhibited for a short period of time by pressure on the carotid sinus (Fig. 30) or by digitalis (Figs. 31 and 32) it is then possible to see the flutter waves undisturbed. The true rapid rate easily becomes apparent and is found to be twice the original ventricular rate. The continuous activity of the auricles is also readily visualized from such tracings.



The rhythm of the auricles is strikingly regular. The ventricular rate and rhythm will depend on the degree of heart block. When there is a 2:1 block, as is commonly the case, the ventricles will necessarily be

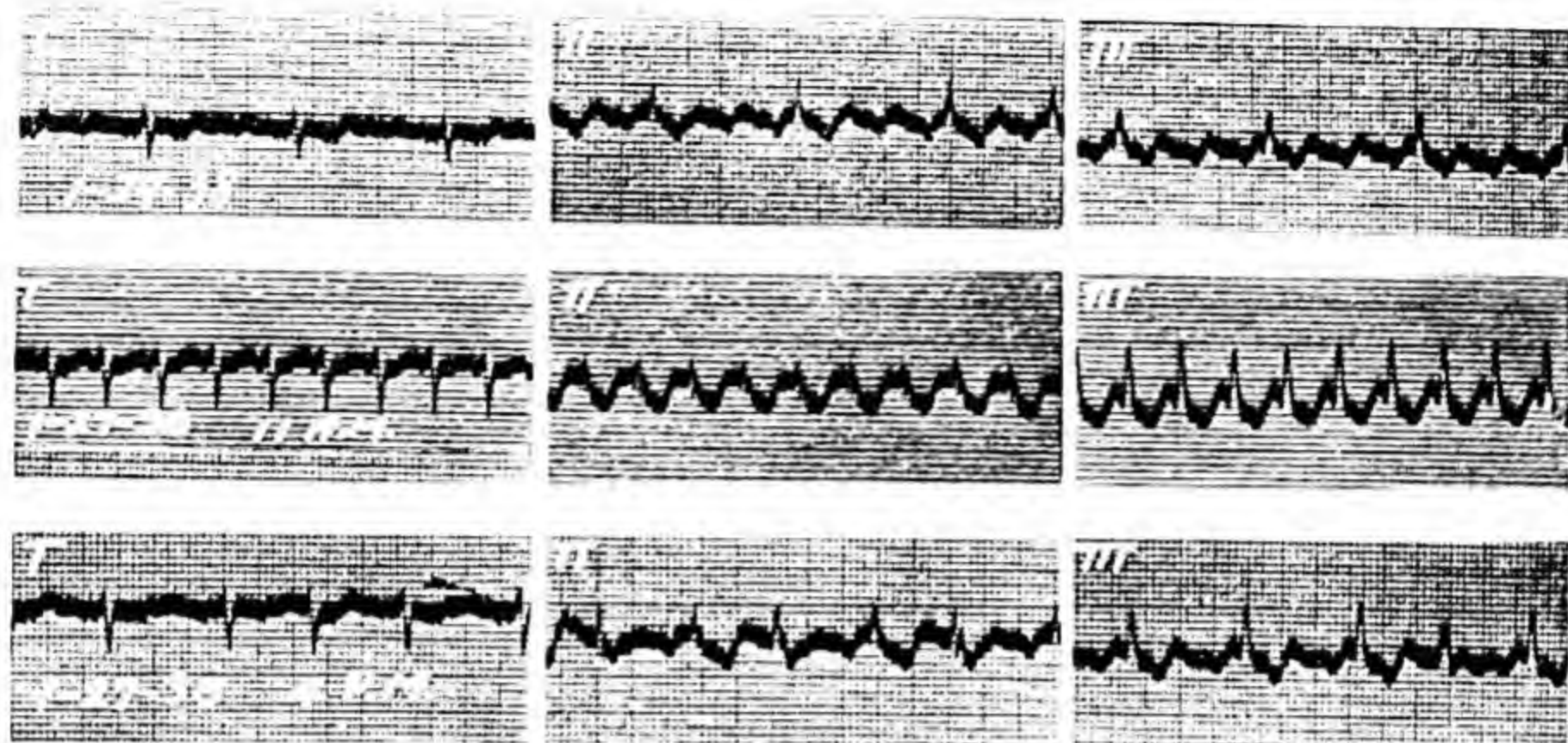


Fig. 33.—Auricular Flutter. Upper curves show mainly a 3:1 block. Middle set shows a 1:1 rhythm. Both auricular and ventricular rates are 191. Lowest set shows a changing degree of block.

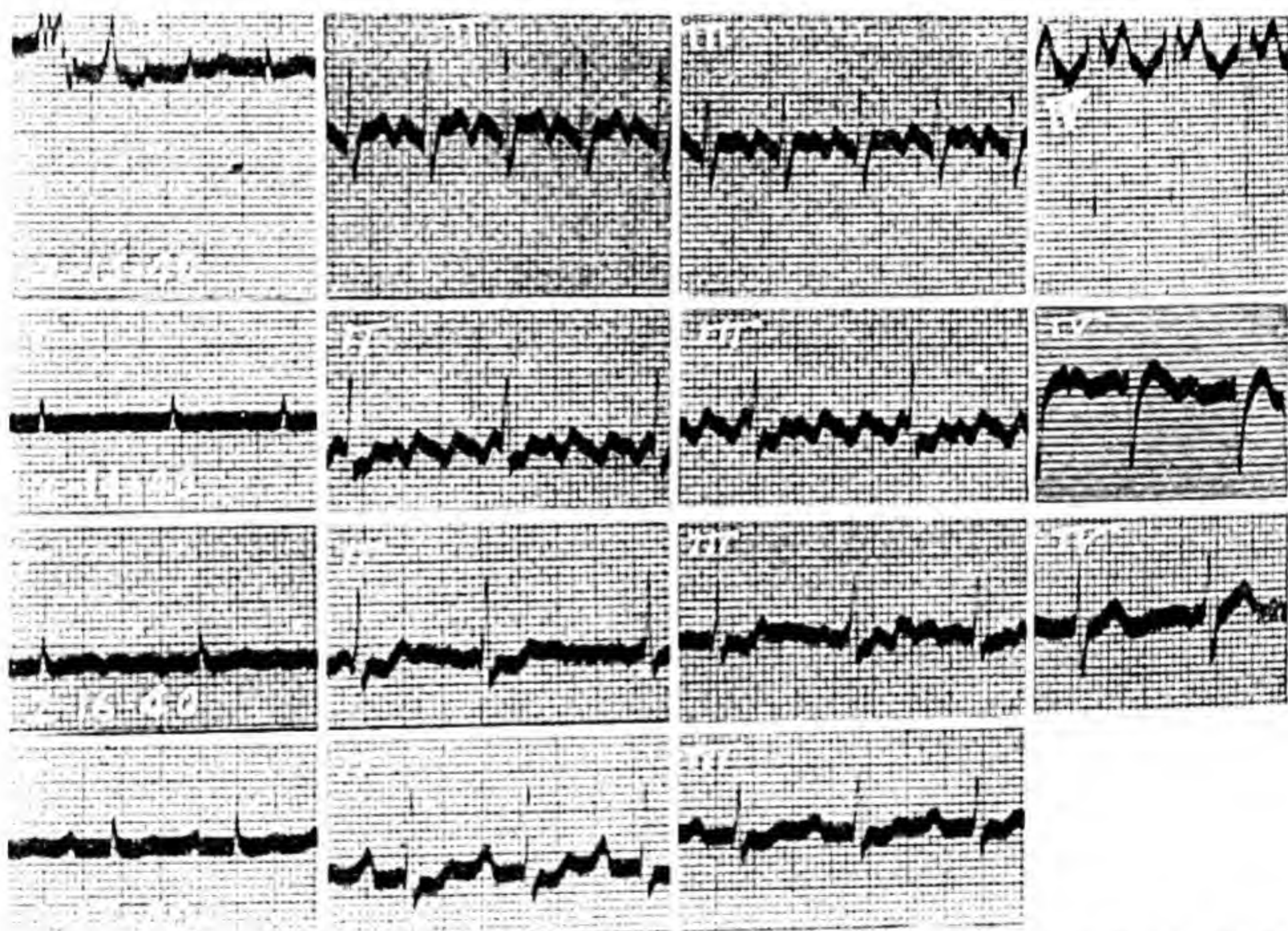


Fig. 34.—Auricular Flutter (Response to Digitalis). Upper set shows auricular flutter with 2:1 rhythm; auricular rate 300, ventricular rate 150; second set shows flutter with 4:1 rhythm, auricular rate 300, ventricular 75; third set shows auricular fibrillation with ventricular rate about 75; lowest set shows normal rhythm rate about 95. The patient received 0.4 gram digitalis orally on February 13, 1940, and 0.6 gram on February 14, 1940, then digitalis was omitted. This patient was sixty-three years old, had mitral stenosis and insufficiency with marked dyspnea and palpitation.

perfectly regular, contiguous cycles not varying more than 0.01 second in length, just as in paroxysmal auricular tachycardia. We now have three conditions that might account for a rapid regular heart rate, *i.e.*,



normal tachycardia, auricular tachycardia and auricular flutter. It often is possible to distinguish these at the bedside. If the regular rate that is counted at the apex is 190 or more, the condition is most likely paroxysmal tachycardia, for a normal tachycardia rarely reaches that high level and the condition is not likely to be auricular flutter because the auricular rate would have to be 380 which is too fast, or 190 which is not only too slow but would necessitate a 1:1 mechanism which occurs but is extremely rare (Fig. 33). I have only rarely seen instances of flutter with auricular rates of 380 to 390, once during a thyroid storm and once during lobar pneumonia. It must be remembered that on auscultation only the ventricular beats are heard, as the auricular are inaudible. Furthermore, if vagal stimulation produced by breathing, carotid pressure or ocular pressure terminates the rapid rate completely the condition must be paroxysmal tachycardia for neither normal tachycardia nor flutter ever respond in this fashion. If the ventricular rhythm is temporarily disturbed and slowed the diagnosis of auricular tachycardia is eliminated for in the latter the tachycardia either would be unaffected or would disappear entirely. When the ventricular rate is slowed, therefore, the condition may still be either flutter or normal acceleration (Figs. 30 and 70). The length of the heart cycles during these interruptions, the method by which the rapid rate is resumed and other factors may then distinguish the one from the other. During the long pauses if frequent auricular waves can be seen in the jugular pulse, auricular flutter is present. Similarly this will be the diagnosis if the heart rate at any time, even if for very short intervals, is found suddenly to be halved. Figure 35 illustrates the response of various types of rapid heart action to vagus stimulation produced by carotid sinus pressure. From such observations one is enabled to distinguish one type from another by simple bedside examination.

When digitalis is given to a patient with auricular flutter the original degree of block increases. Instead of every second, only every fourth beat may reach the ventricles (Fig. 31). The ventricular rate will now necessarily be perfectly regular and slow. If we assume the original auricular rate to have been 300, and the ventricular 150, the auricular rate will be the same but the ventricular 75. When a patient is seen for the first time under such circumstances the underlying mechanism can easily be overlooked for one would not suspect that the rhythm were abnormal with a regular rate of 75. If one thought about the possibility of auricular flutter, one might search for and detect numerous auricular waves in the jugular veins because the auricles are contracting constantly. Of greater help than this is to have the patient exercise a bit. The heart rate may be found to jump suddenly to exactly twice its former rate for a short time, *i.e.*, the original 2:1 block returns, or an irregularity will develop that will lead to the proper diagnosis.

A further effect of digitalis is that the ventricular rate may become irregular. The degree of block then varies. Sometimes every second beat



or every third or fourth beat comes through to the ventricles (Fig. 32). The irregularity may seem to be gross and resemble the total irregularity of auricular fibrillation. On careful examination, however, it will be found that there still remains a dominant rhythm, as the ventricles respond to regularly beating auricles. Grouping of beats will occur that are of equal lengths, inasmuch as they correspond to equal numbers of auricular cycles (Fig. 32). This distinguishes it from auricular fibrillation.

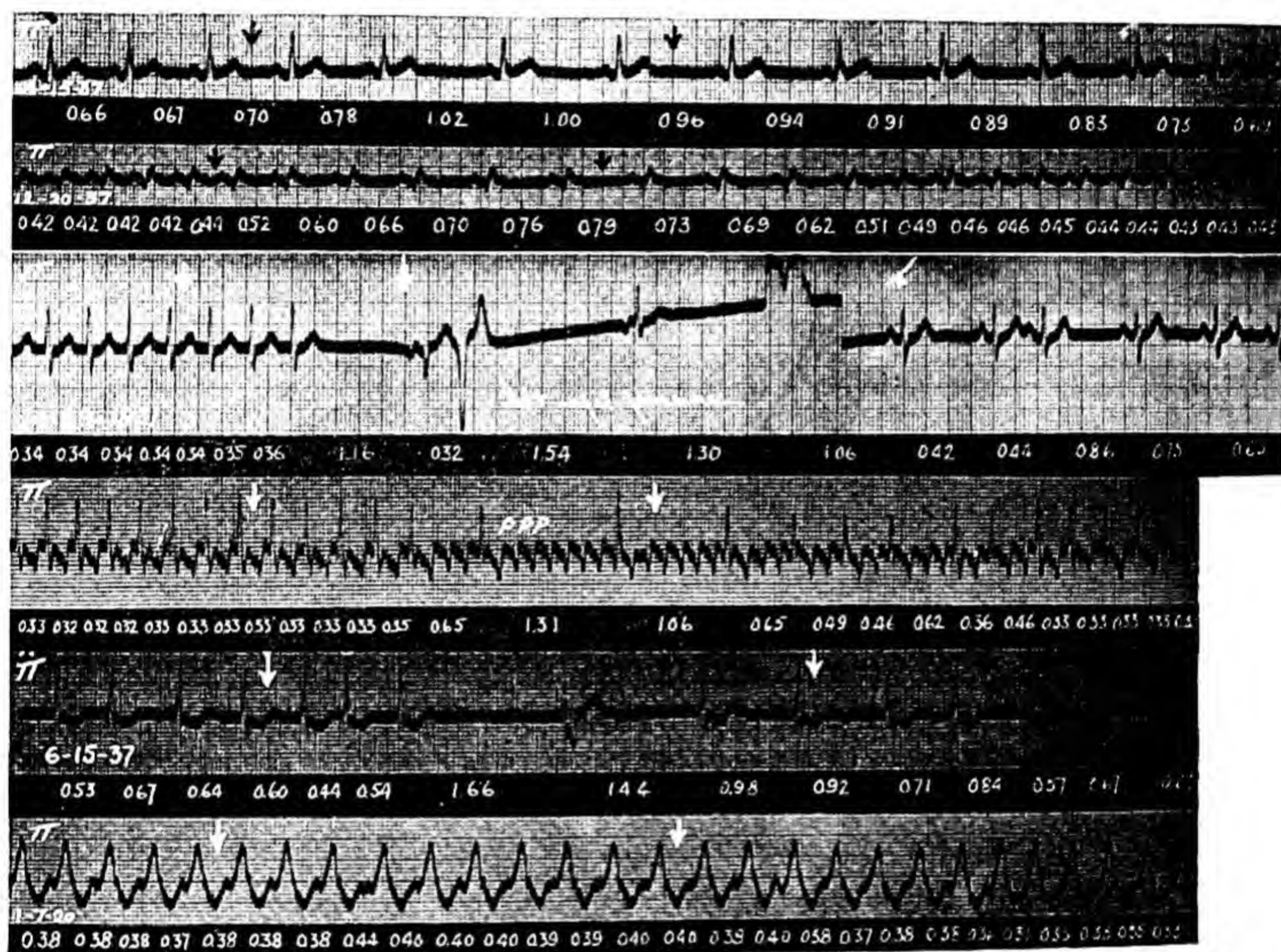


Fig. 35.—Effect of Vagal Stimulation. Arrows in each tracing indicate duration of carotid sinus pressure. Numbers indicate duration in seconds of respective heart beats. First set shows normal gradual slowing in *normal rhythm*. Second set shows gradual slowing with gradual return in *normal tachycardia*. Third set shows instant cessation of an attack of *paroxysmal auricular tachycardia*. Fourth set shows temporary slowing of the ventricles with irregular return to the original rapid rate in *auricular flutter*. Note that the auricular rate remains unaffected. Fifth set shows temporary slowing of the ventricles in *auricular fibrillation*, with return of original grossly irregular rhythm. Last set shows no effect in *ventricular tachycardia*, slight irregularities persisting throughout. These effects characterize each type of mechanism.

Unlike paroxysmal tachycardia which generally occurs in otherwise normal hearts, auricular flutter is apt to be associated with organic heart disease, either valvular or myocardial, although it can manifest itself as a purely functional disturbance. It may occur in the form of paroxysms lasting hours or days and be transient, but more often once established it is permanent unless the patient is treated. It can persist for many years. It occasionally develops during coronary thrombosis, hyperthyroidism, rheumatic fever, pneumonia, pericarditis or other infections, or as a terminal event in chronic nephritis.



The aim in the *treatment* of patients with auricular flutter is to restore the normal rhythm or if this is impossible, to keep the ventricular rate slow. When digitalis is given, in about one third or a half of the cases the following changes will take place. First the ventricular rate slows, the flutter continuing. Then after several days the mechanism changes to auricular fibrillation. If the digitalis is omitted at this point, a normal mechanism may quickly be reestablished (Fig. 34). Often the flutter continues on digitalis therapy with a slow ventricular rate or the fibrillation that develops persists. It is preferable to have the latter rather than the former condition. Quinidine is also a valuable therapeutic agent in auricular flutter. On increasing doses the auricular rate will actually slow, at times to well under 200, while the effect on the ventricles will vary. The ventricular rate may be slowed or accelerated. With the diminished rate of the auricles a larger proportion of the beats may reach the ventricles. Finally the circus motion in the auricles may be broken up and a normal mechanism established. At times large doses of quinidine are necessary to accomplish this, even as much as 1.5 or 2 grams (22 to 30 grains) in a single dose (Fig. 29). It is advisable to try digitalis first, as it is safer and if it does not accomplish the desired effect, it is better to have the patient digitalized before giving quinidine. Patients with auricular flutter receiving drug treatment require careful observation and should be in a hospital where the condition can be followed with frequent electrocardiographic records.

**Auricular Fibrillation.**—The highest degree of auricular disturbance is called auricular fibrillation. In this condition the number of auricular impulses is very great, 350 or more. The mechanism, as has been described, is due to a circus motion in which the path is impure and irregular. The auricles do not actually contract, but rather remain distended in diastole and show fibrillary twitching. There is no constant pathological change in the auricles to explain auricular fibrillation and at present it is regarded as a functional derangement accompanying a variety of states. The condition can be easily reproduced in the experimental animal by faradizing the auricles and I have had the opportunity of seeing it in the living human heart in a patient with adherent pericardium who was undergoing a pericardial operation. The number of auricular impulses is so great that only a portion of them can be conducted through the junctional tissue. Therefore, there is always some degree of heart block associated with auricular fibrillation. The ventricular response is grossly irregular, short cycles and longer cycles occurring without rhyme or reason. The peripheral pulse is necessarily irregular both in time and in force of contraction. In the past it has been called perpetual arrhythmia, absolute or total irregularity, and delirium cordis. In more recent years the true nature of this disorder has been clarified and the term "auricular fibrillation" given to it.

It follows from the preceding description that whatever results from the normal presystolic contraction of the auricles would disappear when



fibrillation develops. In the electrocardiogram the normal P wave will be absent and instead there will be found irregular rapid fibrillary waves (f-f-f) for the most part throughout the cardiac cycle, as the auricular activity is continuous. There will also be a totally irregular ventricular response (Figs. 36 to 42). Inasmuch as the impulses that succeed in

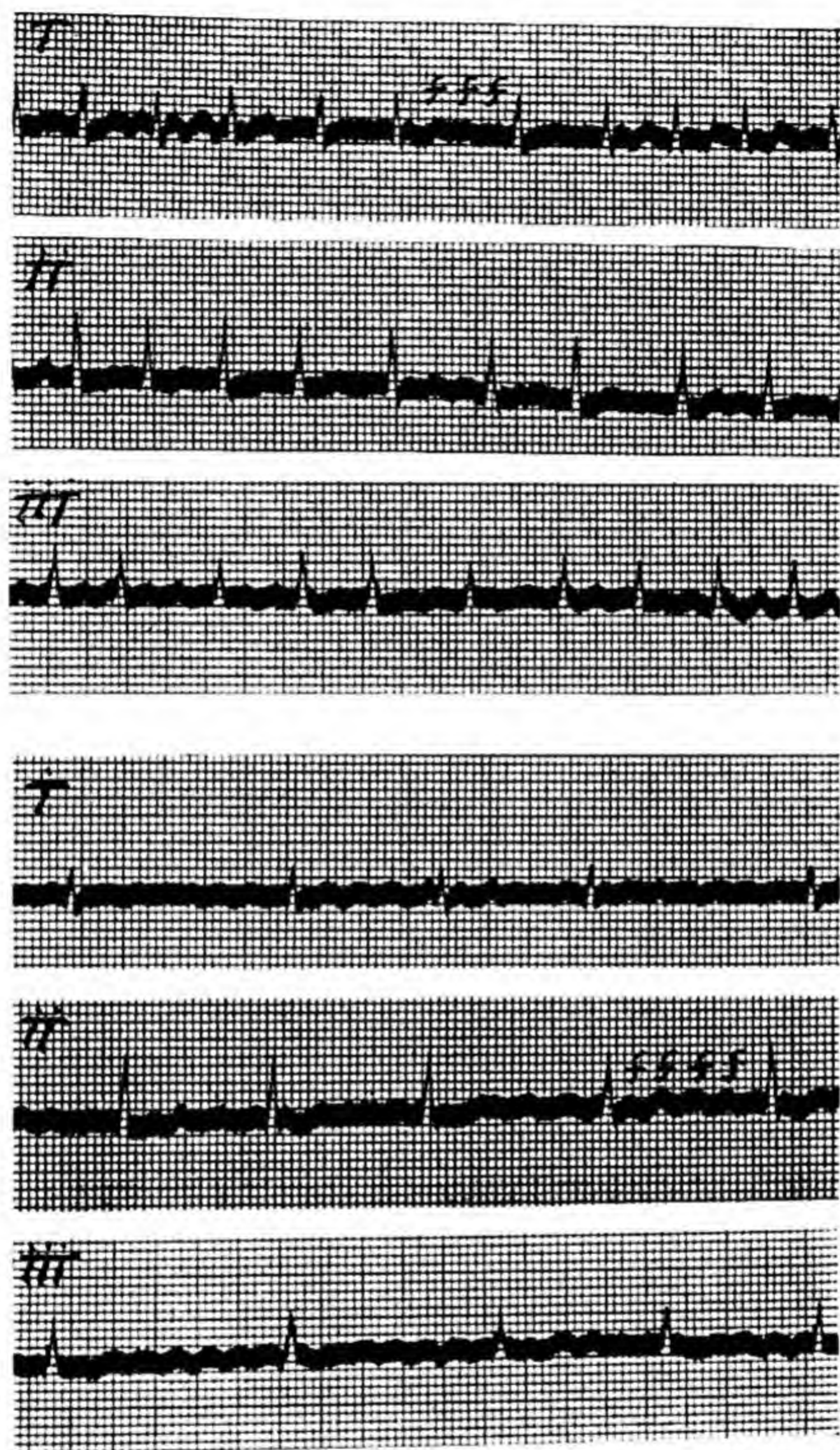


Fig. 36.—Auricular Fibrillation. Effect of Digitalis. The upper three leads were taken July 9, 1935. Note the gross irregularity of the ventricular beats (rate 147), the absence of the normal P wave and the presence of the auricular fibrillary waves (f-f-f), rate 391. The lower three leads taken July 10, 1935 after 1.5 grams of digitalis were given show fibrillation continuing but the ventricular rate is now 60 and still irregular. The patient had mitral stenosis and decompensation.

reaching the ventricles travel down the normal a-v conduction path the ventricular complexes are normal in form. These three features of the electrocardiogram make it quite simple to identify auricular fibrillation, *i.e.*, the absence of the P wave, the presence of fibrillary waves and the gross irregularity of ventricular beats. Sometimes the fibrillary waves



are quite prominent, especially in mitral stenosis, and they may resemble those seen in auricular flutter. It would seem as if the circus motion in places becomes almost perfectly regular. Furthermore, these fibrillary waves may be entirely absent in one or more leads (Fig. 38, Lead III). Occasionally the diastolic interval is perfectly smooth in which case the diagnosis of auricular fibrillation will rest on the other criteria or on taking precordial leads. Finally under certain circumstances, although the auricles are fibrillating, the ventricles contract regularly (Fig. 42). In some cases when digitalis is administered this takes place. It is thought that complete heart block results and while the auricles continue fibrillating the pace is set for the ventricular rate at the a-v node or junctional tissue. When this takes place as a result of digitalis, the regular

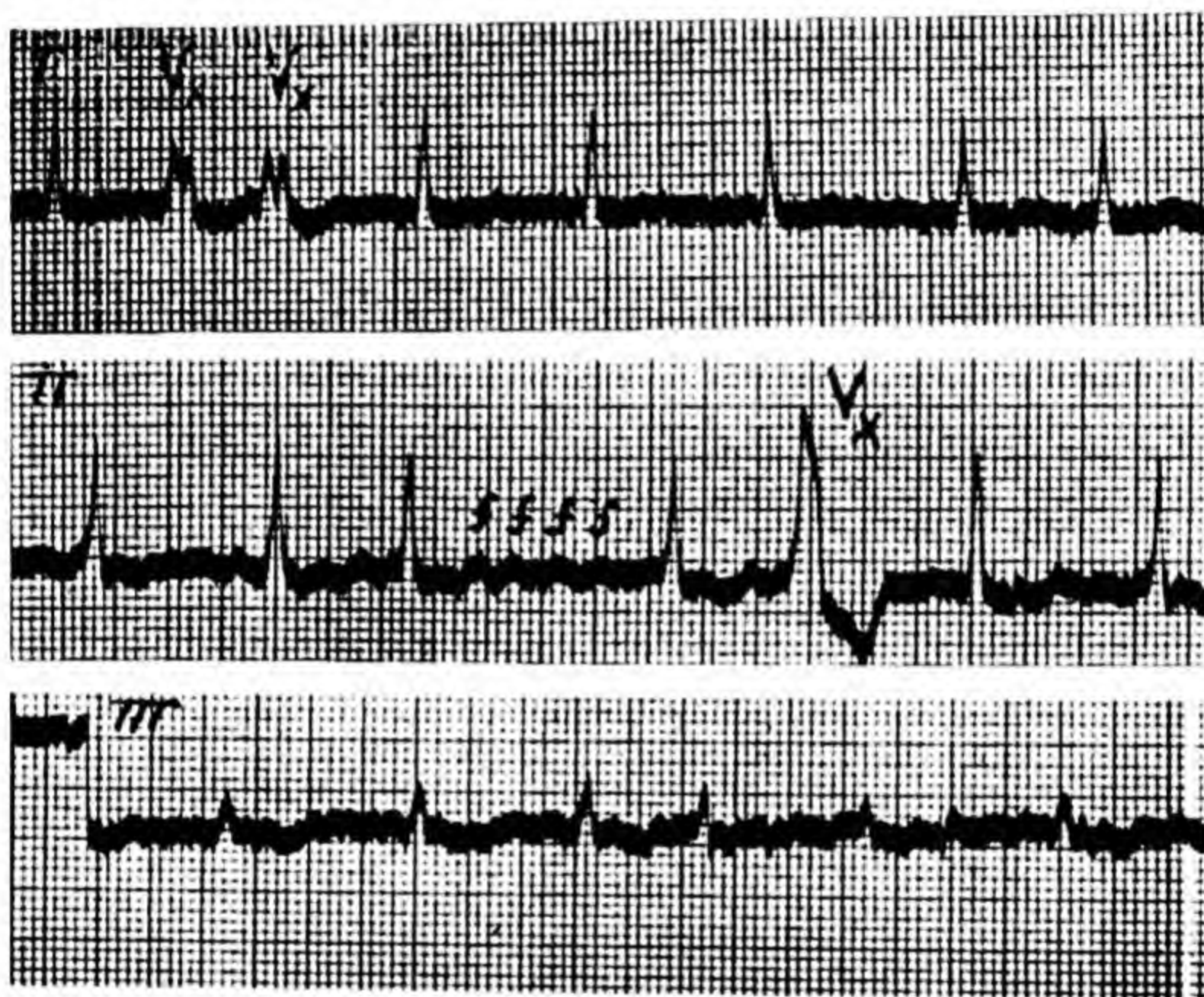


Fig. 37.—Auricular Fibrillation. Note the absolute irregularity of the ventricles, the rapid fibrillation of the auricles (f-f-f) and the absence of the normal P waves.  $V_x$  indicates ventricular extrasystoles. The patient had no heart disease and the rhythm became regular on increasing doses of quinidine.

ventricular rate of complete heart block is not the slow one customarily seen in Adams-Stokes disease. On the contrary the rate will be 55 to 70 or more and on increasing the dose may exceed 100. In fact, if digitalis is continued in large doses a fatal intoxication may result. The important inference from this is that when a patient with auricular fibrillation develops a regular rhythm while taking digitalis the change may be due either to a resumption of the normal rhythm or to the development of this peculiar type of complete heart block. On rare occasions true spontaneous complete heart block with a ventricular rate of 30 may be associated with auricular fibrillation.

From a clinical point of view auricular fibrillation is the most important of all disturbances in the mechanism of the heart beat. It is extremely



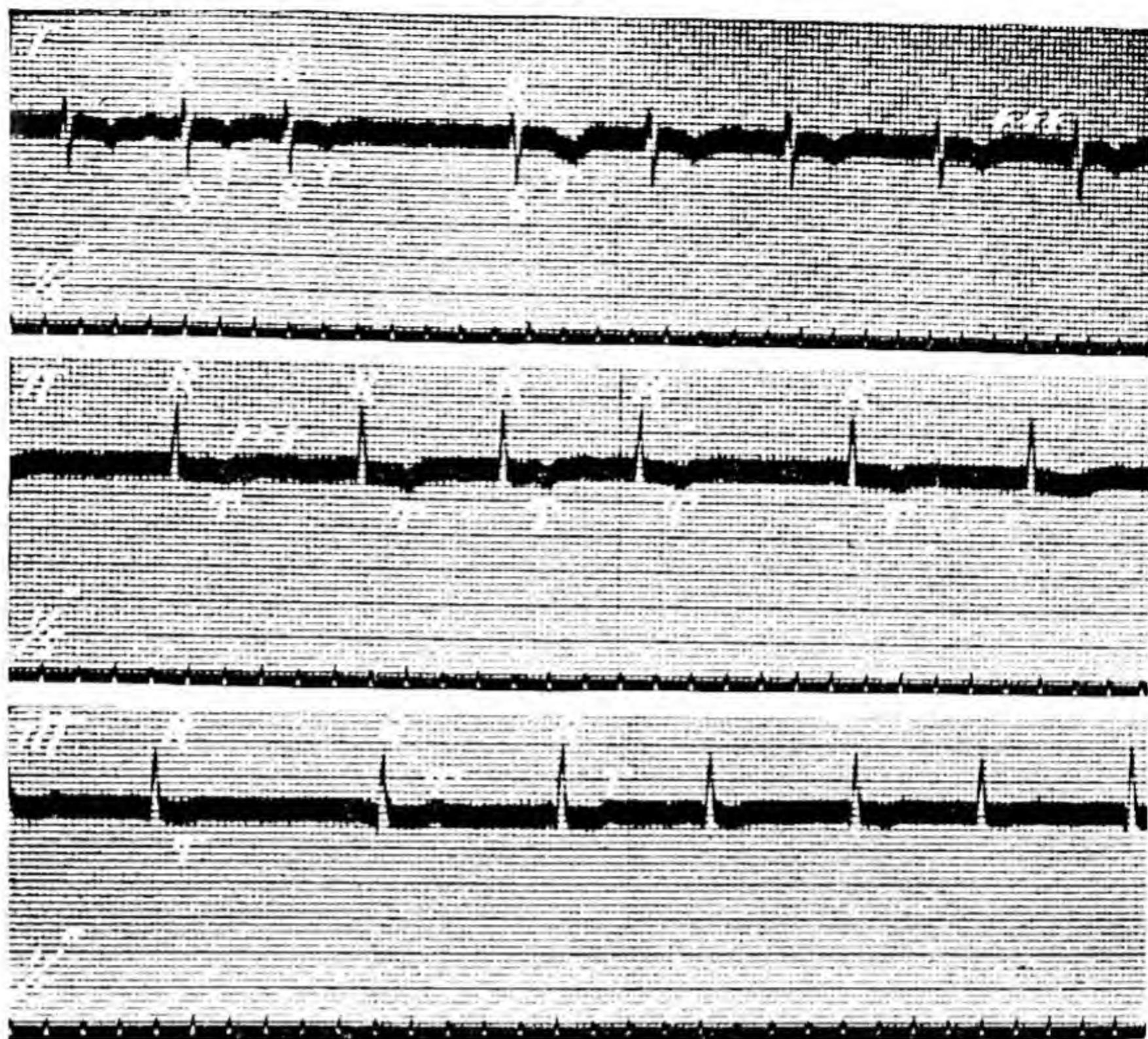


Fig. 38.—Auricular Fibrillation. Note the absolute irregularity of the ventricles and the absence of the P waves. The fibrillatory waves (f-f-f) are practically invisible in Lead III and very small in Leads I and II. The patient had mitral stenosis and decompensation. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

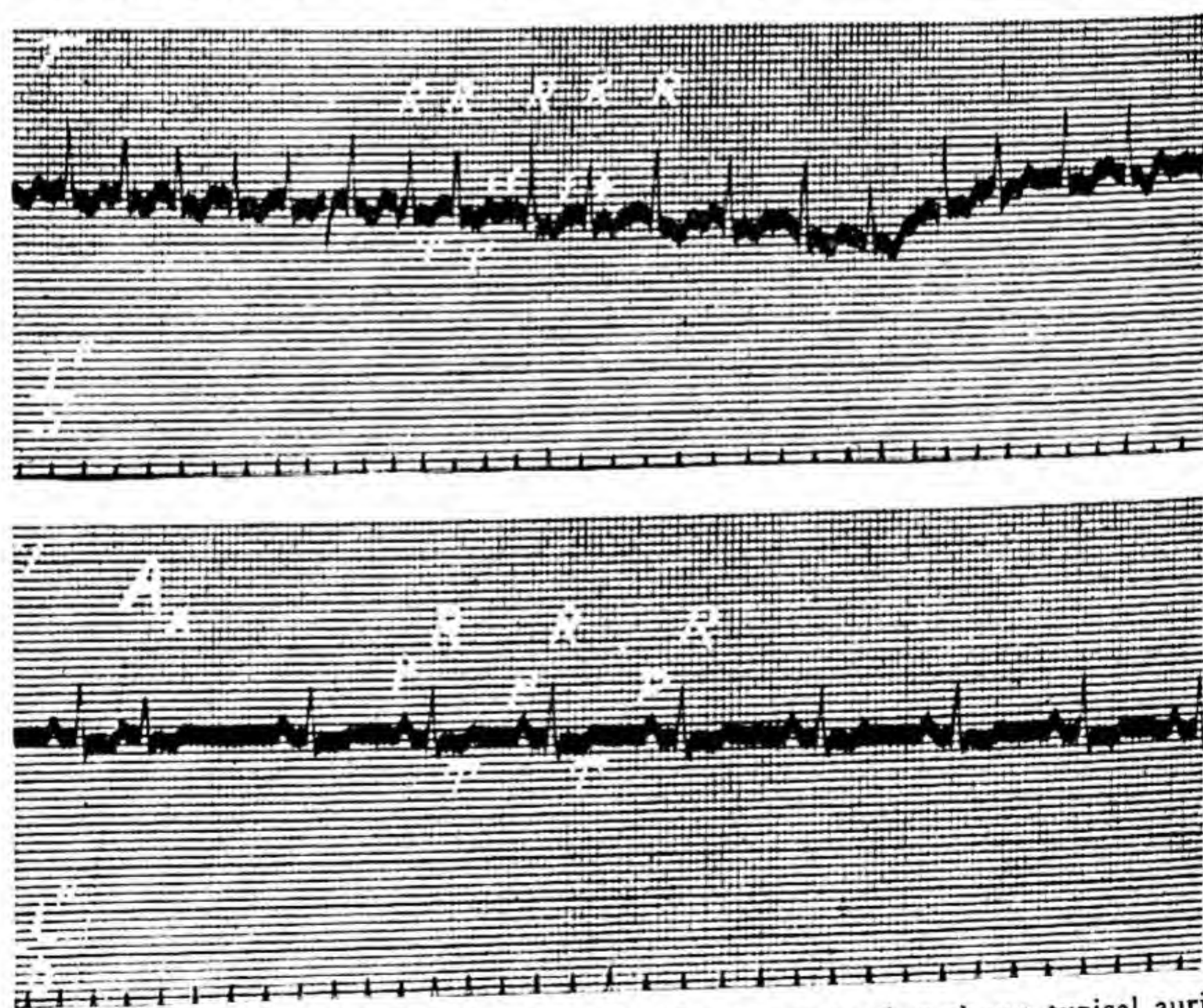


Fig. 39.—Auricular Fibrillation, Paroxysmal. The upper tracing shows typical auricular fibrillation with an irregular ventricular rate of 157. The lower tracing taken a few days later shows normal auricular contractions (P waves), rate 85. There is one premature auricular beat ( $A_x$ ). (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)



common, it produces considerable disability if untreated and responds very satisfactorily to treatment. It occurs in the transient (Fig. 39) as well as in the permanent form. When paroxysms occur they generally last several hours or a day or so. When the irregularity has lasted for

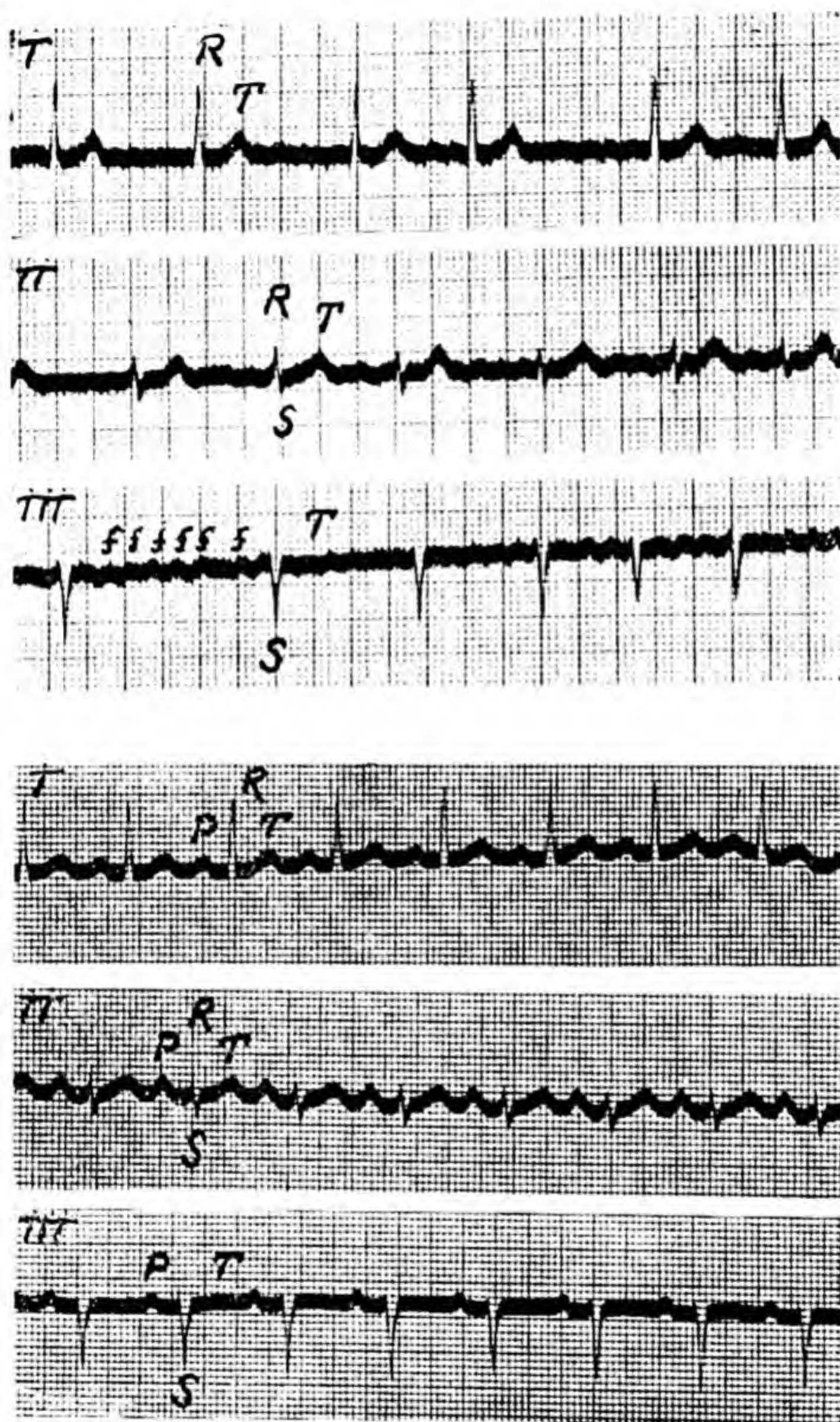


Fig. 40.—Auricular Fibrillation. Effect of Quinidine. Upper three leads taken June 19, 1935 show typical auricular fibrillation. Lower three leads taken June 28, 1935 show a normal rhythm. Increasing dose of quinidine sulfate up to 0.5 gram, three times daily, had been given in the meantime. Contrast this with the effect of digitalis, Fig. 36. This patient had no organic heart disease.

about a week it may be expected to persist indefinitely unless specific measures are taken to restore the normal mechanism. The most common conditions with which it is associated are rheumatic valvular disease with mitral stenosis, hypertensive heart disease, coronary artery disease



and hyperthyroidism. It occasionally develops suddenly during acute infections like pneumonia and rheumatic fever. It is common during the early days following an acute coronary thrombosis. Gallstones are not

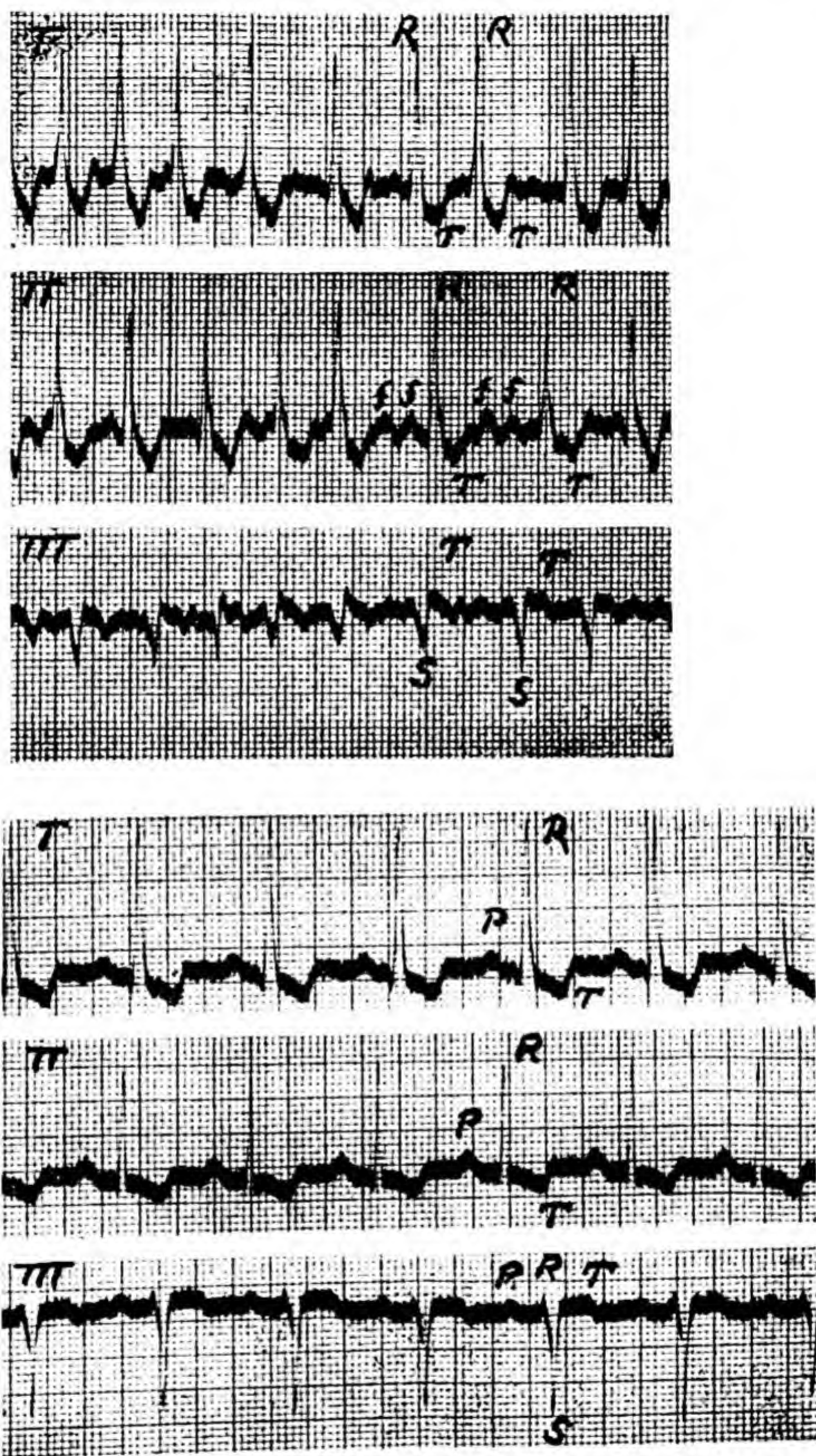


Fig. 41.—Auricular Fibrillation. Effect of Quinidine. Upper three leads are typical of auricular fibrillation; lower three leads show a normal rhythm. Regularization occurred the morning after three doses of 0.3 gram of quinidine sulfate were given. The patient was a man, twenty-five years old, with mitral stenosis and aortic insufficiency.

infrequently found in some of the elderly individuals who have the transient form of auricular fibrillation, but whether there is any association between the two conditions is not certain. On rare occasions auricular fibrillation develops as a result of digitalis therapy. Finally, it may be



present either as a paroxysmal or as a permanent phenomenon in otherwise healthy individuals. This latter group of patients is an important

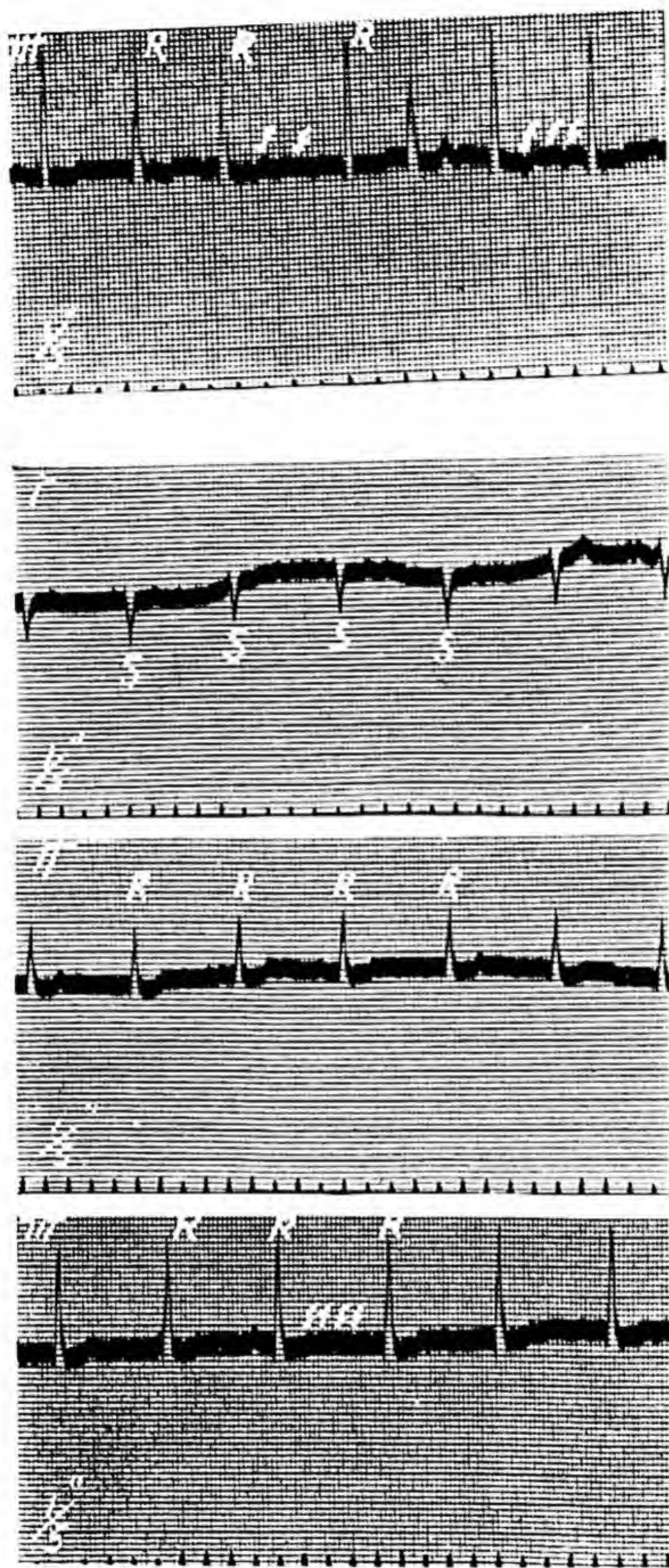


Fig. 42.—Auricular Fibrillation with Regular Ventricular Rhythm. The upper curve shows the typical gross irregularity. The lower three leads were taken after complete digitalization and show that the fibrillation continues although the ventricles are perfectly regular, *i.e.*, probably complete heart block of the toxic type due to digitalis. (Author's article in Oxford Loose-Leaf Medicine vol. II. Courtesy of Oxford University Press.)

one and curiously most of its members are males. Paroxysmal auricular fibrillation is more common than is generally supposed. It is particularly



characteristic of hyperthyroidism and may be the single clue that leads one to this diagnosis. In fact, it is wise to suspect hyperthyroidism whenever a transient spell of this irregularity occurs.

The bedside recognition of this condition is generally simple. A rapid, apparently grossly irregular heart with an appreciable pulse deficit (the count at the apex being 10 beats or more greater than at the wrist) is due to auricular fibrillation nine times out of ten. The common association between a past history of rheumatic fever or chorea, the presence of mitral stenosis, and auricular fibrillation enables one to predict the presence of one of these three factors if the other two are known to exist. If a patient had rheumatic fever and shows signs of mitral stenosis, then a grossly irregular heart is almost always due to auricular fibrillation. If this irregularity is present and there is a history of rheumatic fever, there probably is mitral stenosis. Finally, if the patient has mitral stenosis and auricular fibrillation, he has been rheumatic whether a positive history can be obtained or not. Exceptions will be found for these generalizations but it is surprising how frequently they will be valid.

When auricular fibrillation first develops the ventricular rate is quite rapid except in rare instances in which some defect in the junctional tissue already is present. When digitalis is given it slows the ventricular response. Figure 36 shows such an effect, the ventricular rate falling from 147 to about 60 in twenty-four hours after a large dose of digitalis was administered. It must be borne in mind that the fibrillation of the auricles persists. Digitalis does not stop the fibrillation; it merely prevents many of these rapid beats from reaching the ventricle through its vagal influence on the conduction tissue. Physicians are too inclined to use the expression that "the fibrillation is less" when what is meant is that ventricles are slower or less tumultuous. The fibrillation of the auricles is either present or absent; from a practical point of view there are no degrees of fibrillation. When the ventricular rate slows, however, the pulse deficit diminishes and may entirely disappear. At this time the bedside rule of thumb is no longer diagnostic. If a physician first sees a patient under these circumstances, the diagnosis is more difficult and must be made on other criteria. Irregularities of the heart beat and of the pulse, though slight, will still be present. The condition may resemble other arrhythmias. Exercise may accelerate the rate and make the more characteristic features return. Rarely will it be necessary to have electrocardiograms before a final decision can be made. There is one additional clinical finding that may help to distinguish fibrillation from a very irregular rhythm due to numerous extrasystoles. In both conditions cycles of short, long or medium duration may occur at various times. In irregular rhythm due to extrasystoles it will be found that with extrasystoles all the long pauses are preceded by quick beats because they are compensatory. The same auscultatory phenomenon is present with auricular fibrillation. But if one listens long enough and hears a sudden pause not preceded by a quick beat, this points to auricular fibrillation. (See the



fifth heart cycle in Lead II, Fig. 38.) In other words, a sudden lengthening of the heart cycle after a beat of normal length is more characteristic of auricular fibrillation than is a sudden shortening of the cycle.

The customary *treatment* for patients with auricular fibrillation is digitalis. The purpose is to slow the ventricular rate and if this slowing does not result from adequate dosage, one may rightly suspect the drug is not sufficiently potent or that the patient has hyperthyroidism. Ordinarily the slowing is marked and may be compared in its specific effect to the action of quinine in malaria. Furthermore, no similar slowing can be expected from digitalis on a normal tachycardia. This explains why some cardiac patients improve after fibrillation develops, for the digitalis that had been given previously without effect only then begins to slow the rate.

Quinidine, on the other hand, is used at times to treat patients with auricular fibrillation. (See Chapter 20.) It is used less than formerly but occasionally is a very valuable aid to our treatment. The purpose here is to restore the heart to a regular rhythm (Figs. 40, 41). It does so by

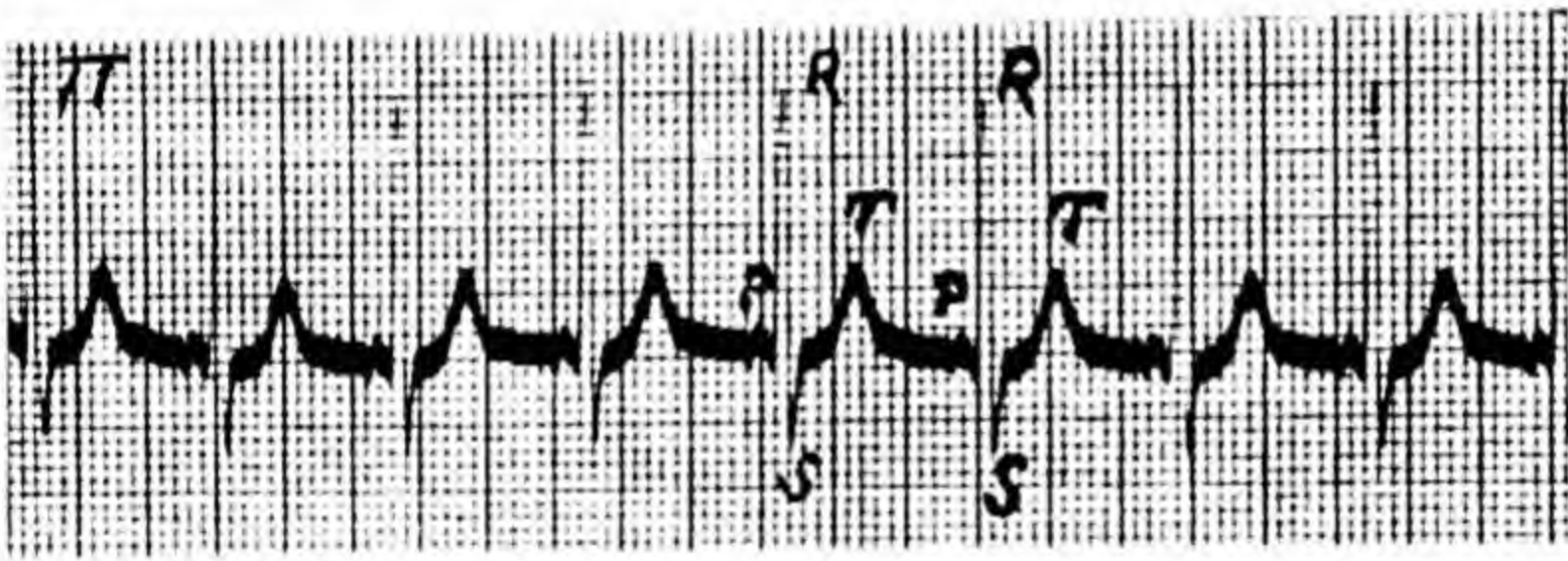


Fig. 43.—Nodal Rhythm. Note that the P waves have a peculiar form and that the P-R interval is very short (0.06 second). The ventricular complexes are normal. This patient had acute rheumatic carditis.

lengthening the refractory period of the auricular musculature so that when the wave completes its circuit it finds the tissue ahead of it still refractory and the circus ends, permitting the normal pacemaker to send out its impulse and reestablish ascendancy over the beat of the heart. The reason that quinidine does not always break up fibrillation is that it also slows the speed of the impulse, which would tend to perpetuate the circus motion by allowing sufficient time for tissue to recover from its retractoriness. When the first effect predominates fibrillation ceases, when the latter predominates it continues. Quinidine is particularly useful in restoring normal rhythm if there is no organic heart disease and in a few other selected cases of auricular fibrillation.

**Ectopic Auriculoventricular (Nodal) Beats.**—Abnormal beats may arise in the auriculoventricular node or in the bundle of His. The pacemaker in the junctional tissue may control the rhythm of the heart for long periods of time (Figs. 43, 44) or only isolated beats may arise (Fig. 45). The impulse will travel down the ventricles through normal pathways and therefore the ventricular complexes will be normal. It



reaches the auricles, however, in a reversed direction and so will produce abnormal P waves. The auricular contraction may take place shortly before or after the ventricular depending on which receives the impulse first. If the focus is near the top or auricular end of the node, it will reach the auricles quickly, but the P-R interval will be shorter than normal. If

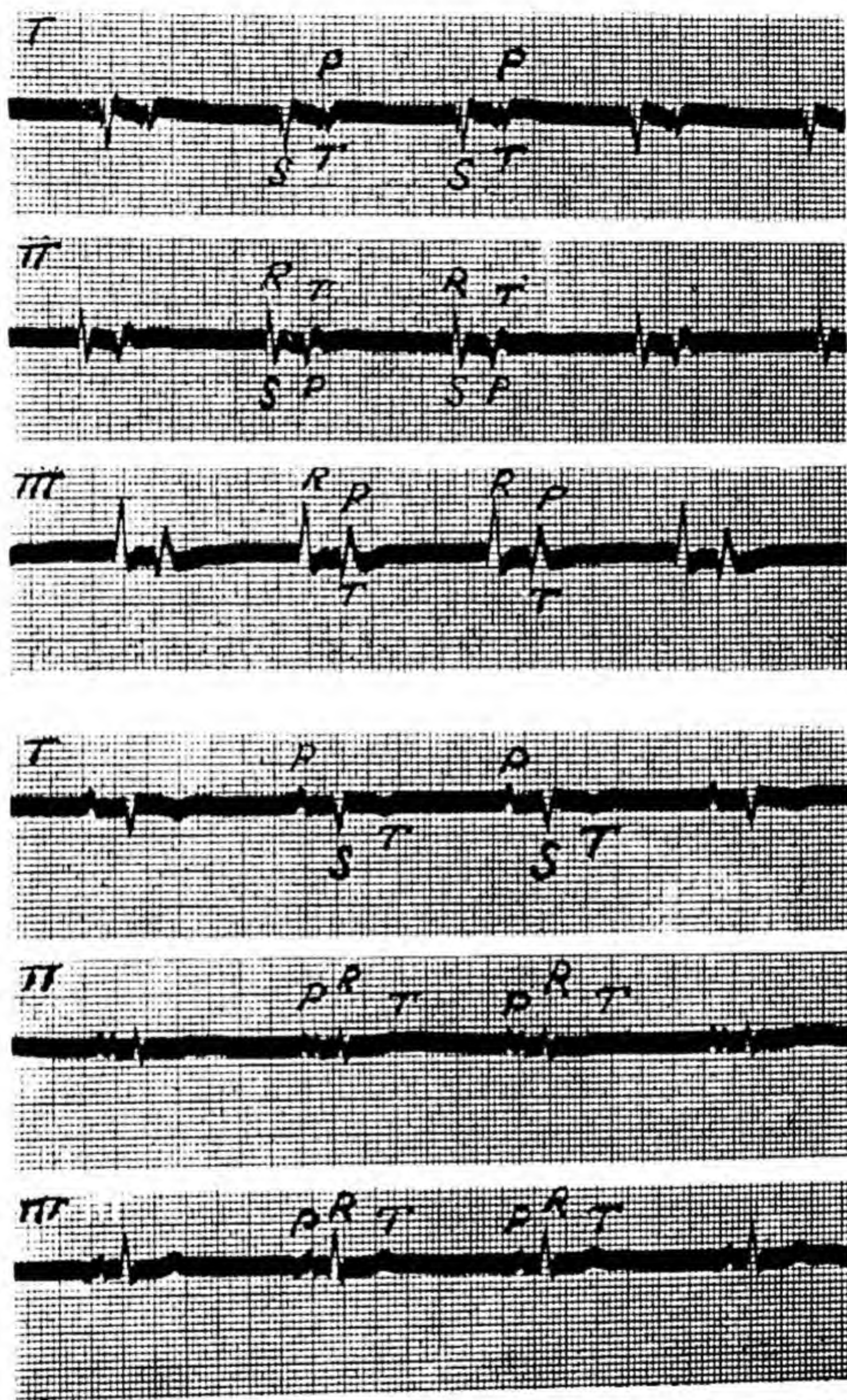


Fig. 44.—Nodal Rhythm. The upper three leads show P waves of a peculiar form coming at a constant interval after the R waves (R-P interval of 0.23 second). The lower three leads show a return to the normal rhythm with a P-R interval of 0.22 second. The patient had alcoholic cirrhosis of liver.

the focus is lower down, the ventricles will respond before the auricles and an R-P rather than a P-R interval results (Fig. 44). At times within the same tracing varying positions of the P wave will be found, sometimes before or after or even simultaneously with the R wave. The normal beat from the sino-auricular node may produce auricular contraction



Nodal rhythms have no great clinical significance and cannot be identified without graphic methods. They may occur during anesthesia, deep breathing or vagal stimulation and can be produced by certain drugs. There are no practical diagnostic, prognostic or therapeutic problems involved in this type of irregularity.

Although nodal beats cannot be accurately identified without graphic methods, when a series of such beats arise and the relation of auricular and ventricular systole keeps changing, one may suspect the presence of this type of arrhythmia by detecting a changing quality of intensity of the first heart sound. This alteration is the result of the differences in the position of the auriculoventricular valves at the moment ventricular

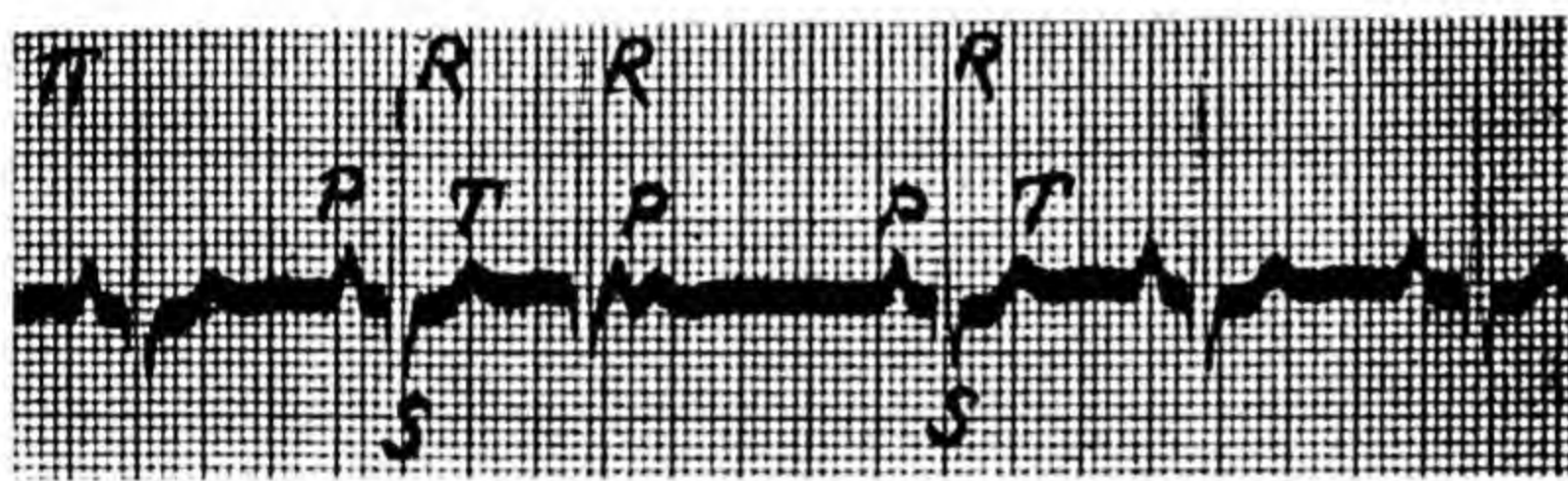


Fig. 45.—Premature Nodal Beat. Note that the premature beat which arises in the a-v node produces a ventricular complex of normal form. The auricular rhythm is undisturbed as the P wave comes exactly on time. There was no heart disease here.

systole occurs. A similar mechanism accounts for variations in the quality of the first heart sound in other arrhythmias, for this sound is mainly, if not entirely, due to the snap of the a-v valves.

**Paroxysmal Nodal Tachycardia.**—One would presuppose that a paroxysm of tachycardia might arise from the a-v node or junctional tissue just as it does from the auricles or ventricles. It is difficult to be certain of this for when such a tachycardia displays abnormal P waves it cannot be determined whether such waves are the result of impulses arising in the auricles, in which case it really indicates paroxysmal auricular tachycardia, or whether they come from the a-v node. It is almost impossible to identify which ventricular complex is related to the particular P wave, (Fig. 46). From a clinical point of view whatever has been said about paroxysmal auricular tachycardia applies to the condition that might be called "paroxysmal nodal tachycardia."

**Ectopic Ventricular Beats.**—Extrasystoles arising in some ectopic focus in the ventricle are extremely common. This arrhythmia is generally the cause of what the physician calls an "intermittent pulse." This latter expression should be given up because intermittence of the peripheral pulse can be due to a variety of disturbances of the heart beat, which



can be properly diagnosed on auscultation of the heart. In this condition the heart is beating regularly, when suddenly a quick beat is heard followed by a pause. The premature beat is the extrasystole and may occur so early in the previous diastole that the pressure and volume of blood

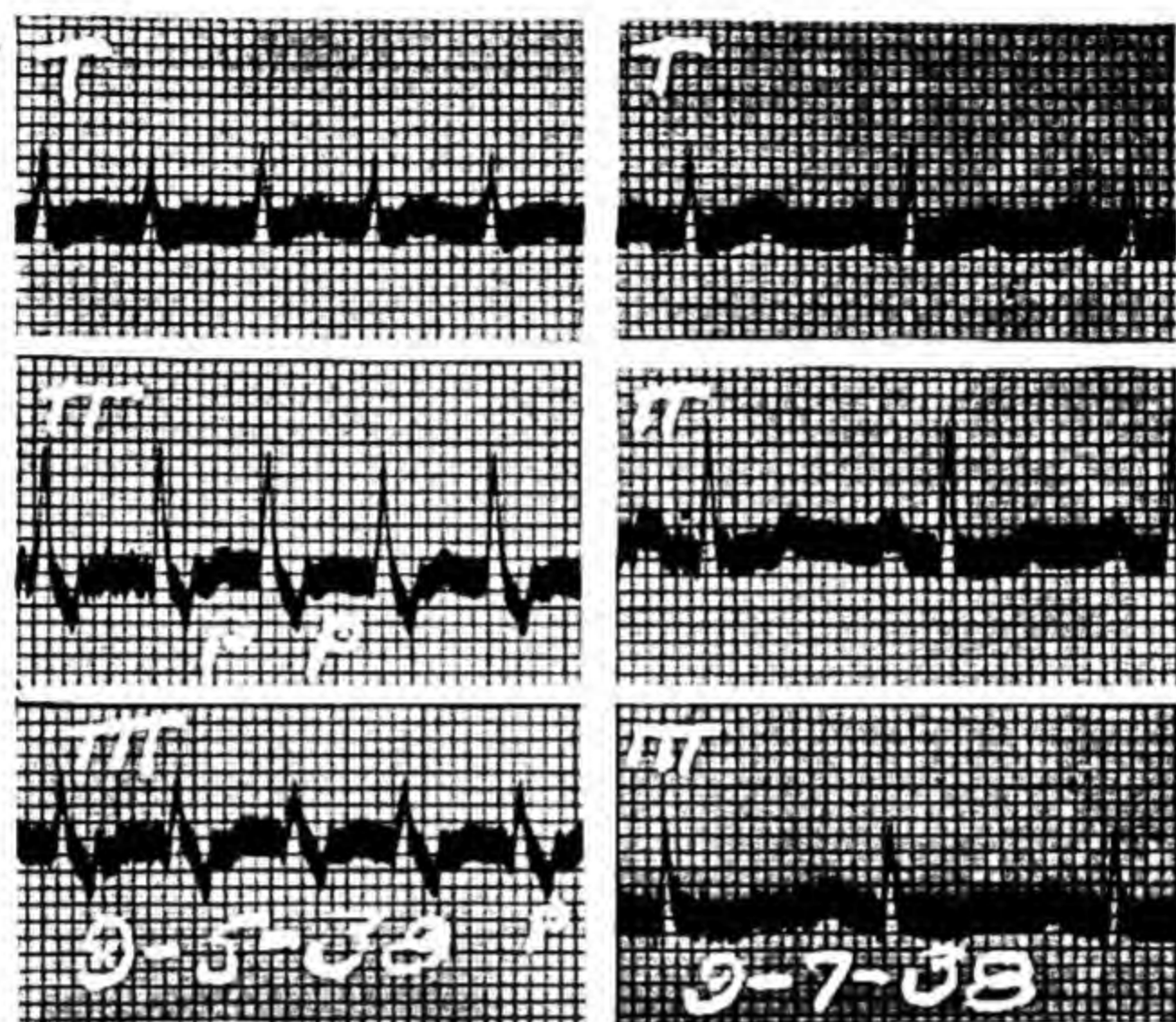


Fig. 46.—Paroxysmal Nodal Tachycardia (Probable). The first set shows regular rate of 201. Note that P waves seem to occur at the R-T junction. The second set shows normal slow rhythm. Some of these attacks were stopped by carotid sinus pressure. The patient was a man fifty-four years old with tubercular constrictive pericarditis and obliterative pleuritis.

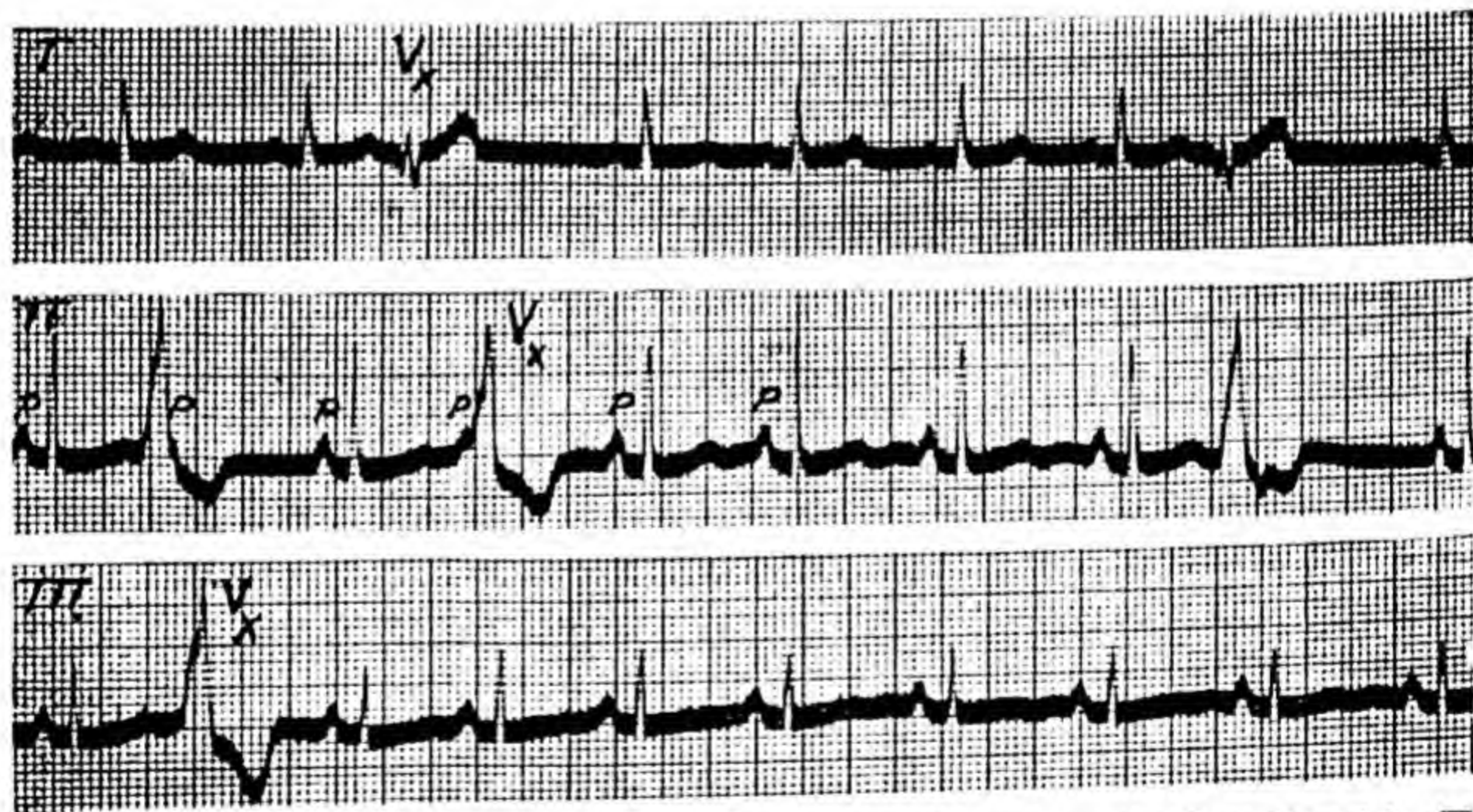


Fig. 47 —Premature Ventricular Beats, Probably Arising from the Left Ventricle. The normal rhythm is occasionally interrupted by ventricular complexes having a decidedly abnormal form ( $V_x$ ). Note that the regularity of the auricular beats (P) is not interrupted although some are buried with the extrasystole. The patient had neurocirculatory asthenia.

in the ventricles often are not great enough to produce a pulse at the wrist (Fig. 52) and occasionally may not even open the aortic valves. In the latter case only one heart sound may be heard over the precordium for that particular beat. I have even observed instances in which ventricular extrasystoles would be seen to occur while electrocardiograms



were being taken which were entirely inaudible during auscultation that was performed simultaneously.

The spread of the impulse from the ectopic focus through the ventricle is abnormal and so the ventricular complex will be bizarre (Figs. 47 to 54). The direction the waves will take will depend on whether the impulse arises in the left or right ventricle and in the basal or apical region. In general the complexes are broad, coarsely notched and the T waves extend in the opposite direction to the main initial ventricular deflections. Inasmuch as the ventricle contracts prematurely, the normal auric-

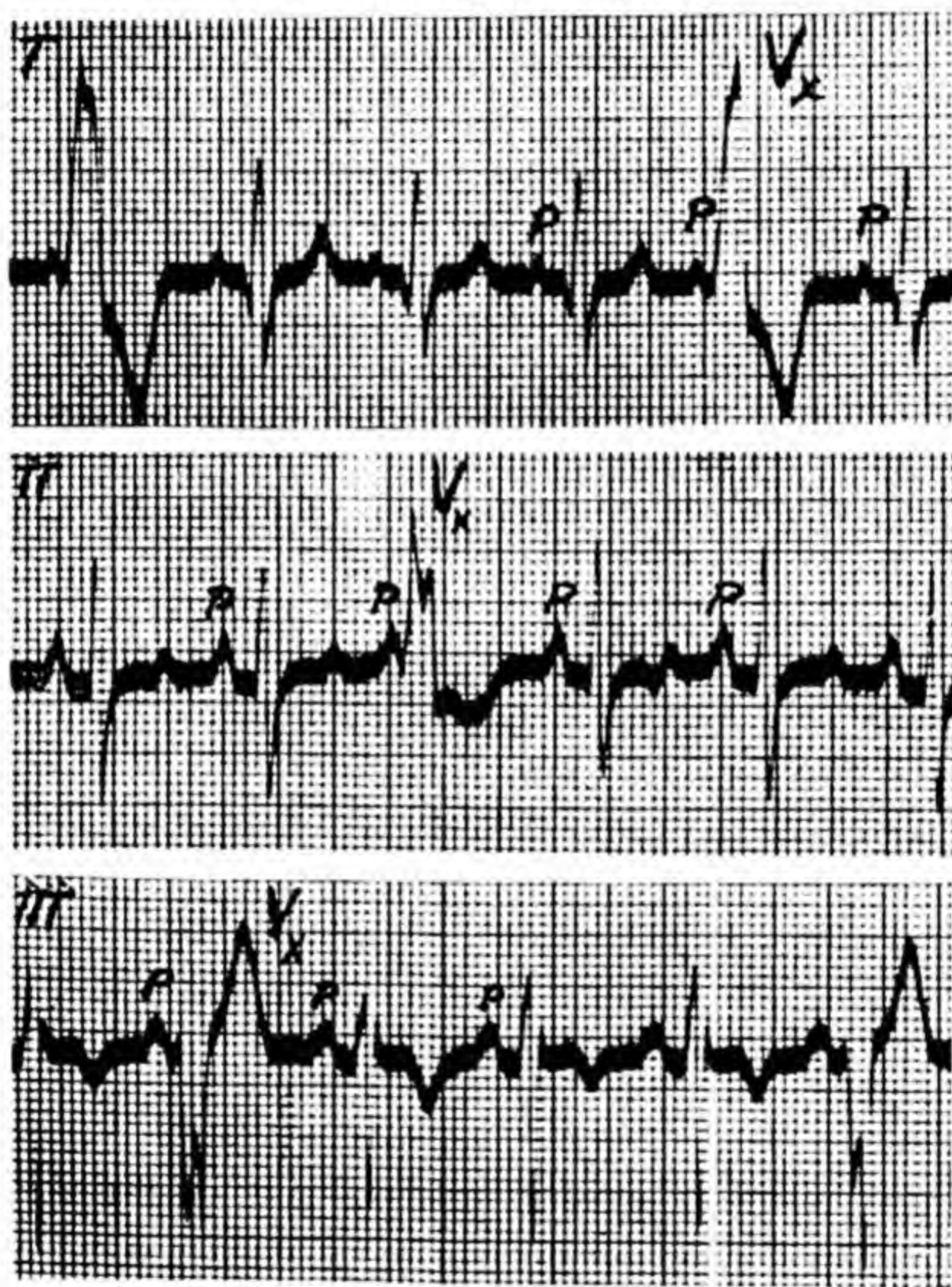


Fig. 48.—Premature Ventricular Beats, Probably Arising from the Right Ventricle. Note the extrasystoles with abnormal ventricular complexes ( $V_x$ ). They are only very slightly premature so that they actually follow the P wave. This patient had congenital heart disease.

ular beat coming down about the same time finds the ventricle refractory so that it cannot respond again. There follows then a pause until the next normal auricular beat. This pause is completely compensatory for the rhythm of the pacemaker is not disturbed and the duration of the two beats including the extrasystole is equal to two normal heart cycles (Fig. 49, Lead II). The P wave is often lost in the ventricular complex (Fig. 49) or may appear before or after the QRS waves (Figs. 47 and 48).

These beats may occur very rarely so that the physician is unable to detect them at the time of his examination or they may be frequent.



They may come every second beat (bigeminy) (Fig. 51) or every third beat (trigeminy) (Fig. 49). Sometimes they come in succession so that there may be a run of two or more extrasystoles producing curious types

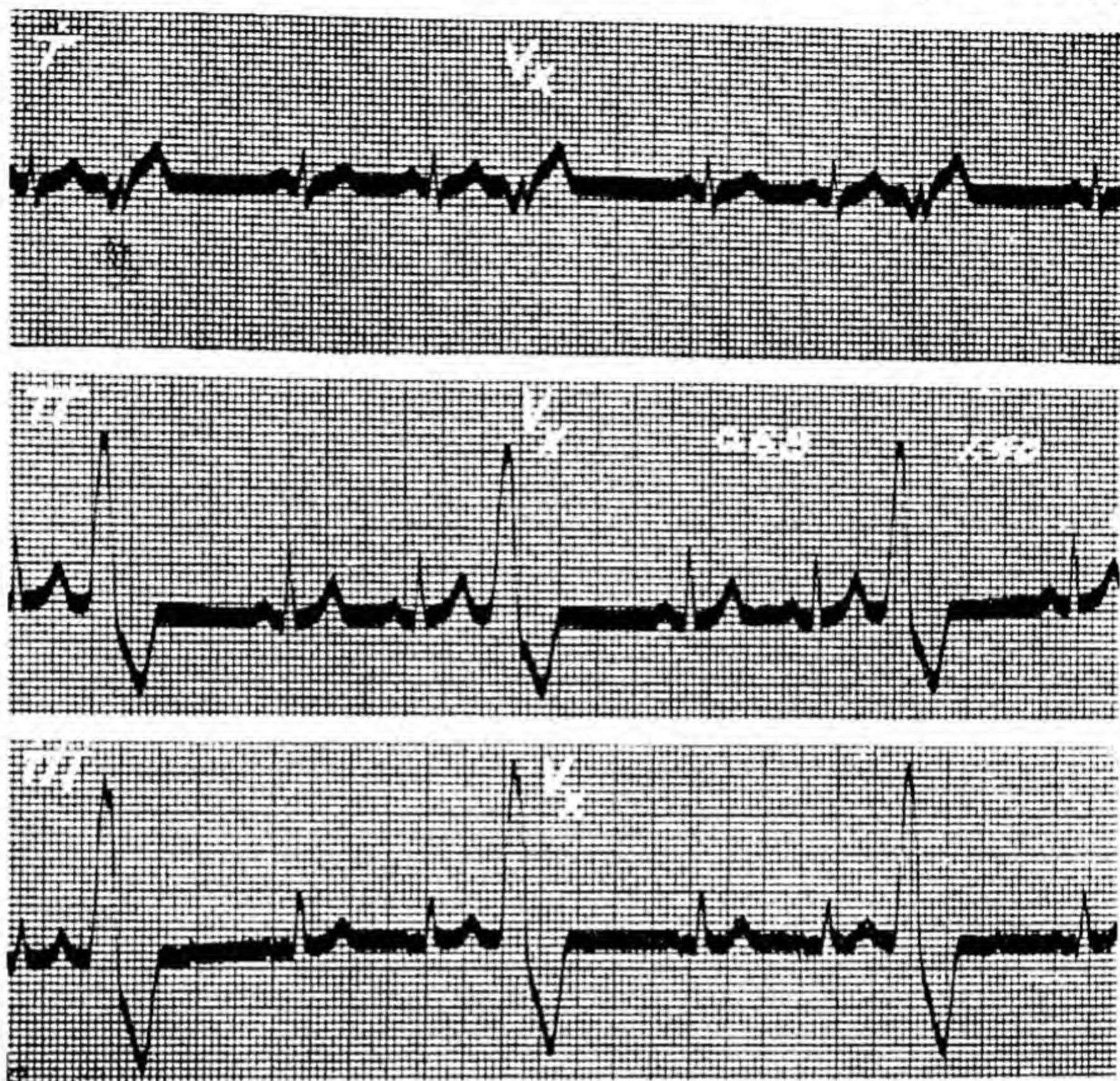


Fig. 49.—Premature Ventricular Beats. Trigeminy. Note the typical ventricular extrasystoles ( $V_x$ ) coming regularly after two normal beats. This patient had no organic heart disease. The length of normal cycle is 0.69 second. The length of the two beats including the extrasystole is just twice the normal beat, 1.40 seconds. Therefore the pause is completely compensatory.



Fig. 50.—Premature Ventricular Beats. Trigeminy. Note that after each normal beat there are two consecutive extrasystoles ( $V_x$ ) followed by a compensatory pause. The auricles (P) continue regularly. The patient had rheumatic valvular disease. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

of irregularity (Fig. 50). When they arise in different parts of the ventricles they will have various forms (Fig. 54). Such a condition is apt to be associated with serious heart disease.



Occasionally ventricular extrasystoles are interpolated between two normal beats. The heart is beating so slowly and the premature beat

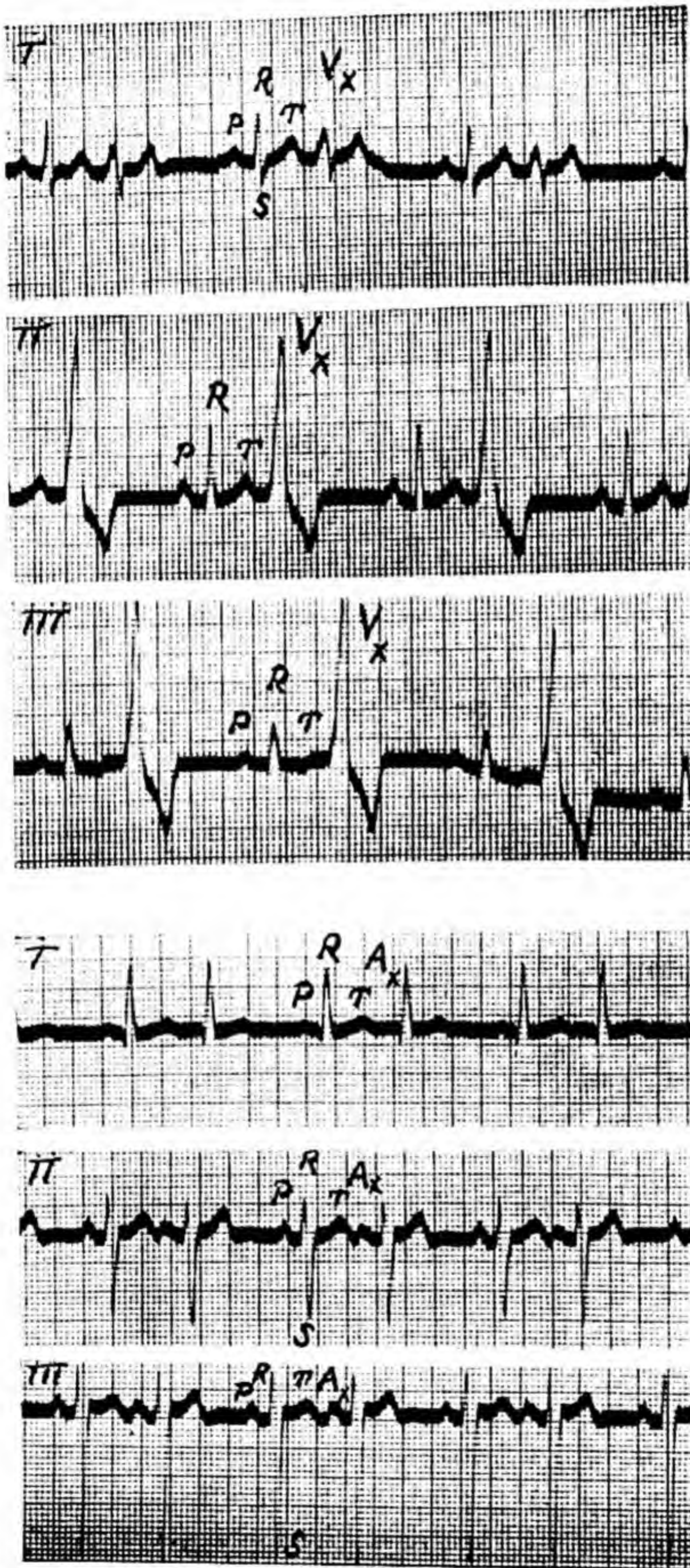


Fig. 51.—Premature Ventricular Beats. Bigeminy. The upper three leads show a ventricular extrasystole ( $V_x$ ) occurring after each normal beat and the lower three show an auricular extrasystole ( $A_x$ ) producing in both cases a coupled rhythm. Note the abnormal form of the ventricular complexes above and the normal ventricular complexes below with auricular waves of abnormal form. Organic heart disease was present in neither case.

occurs so early in diastole that the next normal auricular impulse finds the conduction tissue and the ventricles ready to respond (Fig. 55).



There is no compensatory pause and instead one hears three rapid beats, the second one of which is the extrasystole. Another possible explanation of this mechanism is that the impulse from the ventricles travels through the junctional tissue to the auricles in a retrograde fashion and starts a new impulse at the normal pacemaker.

Ventricular extrasystoles occur very frequently in individuals otherwise normal. They are also common in those having organic heart disease, but the diagnosis of structural disease of the heart will have to rest on other criteria than the irregularity. When they are numerous and the patient requires digitalis for heart failure they indicate a grave outlook for it is more difficult to administer as large doses of the drug as would otherwise be possible. In fact, digitalis in large doses often produces ventricular extrasystoles, especially in the form of digitalis coupling (Figs. 52, 53), even if none were present before the drug was used. Coupling may occur whether the rhythm was previously normal, in which

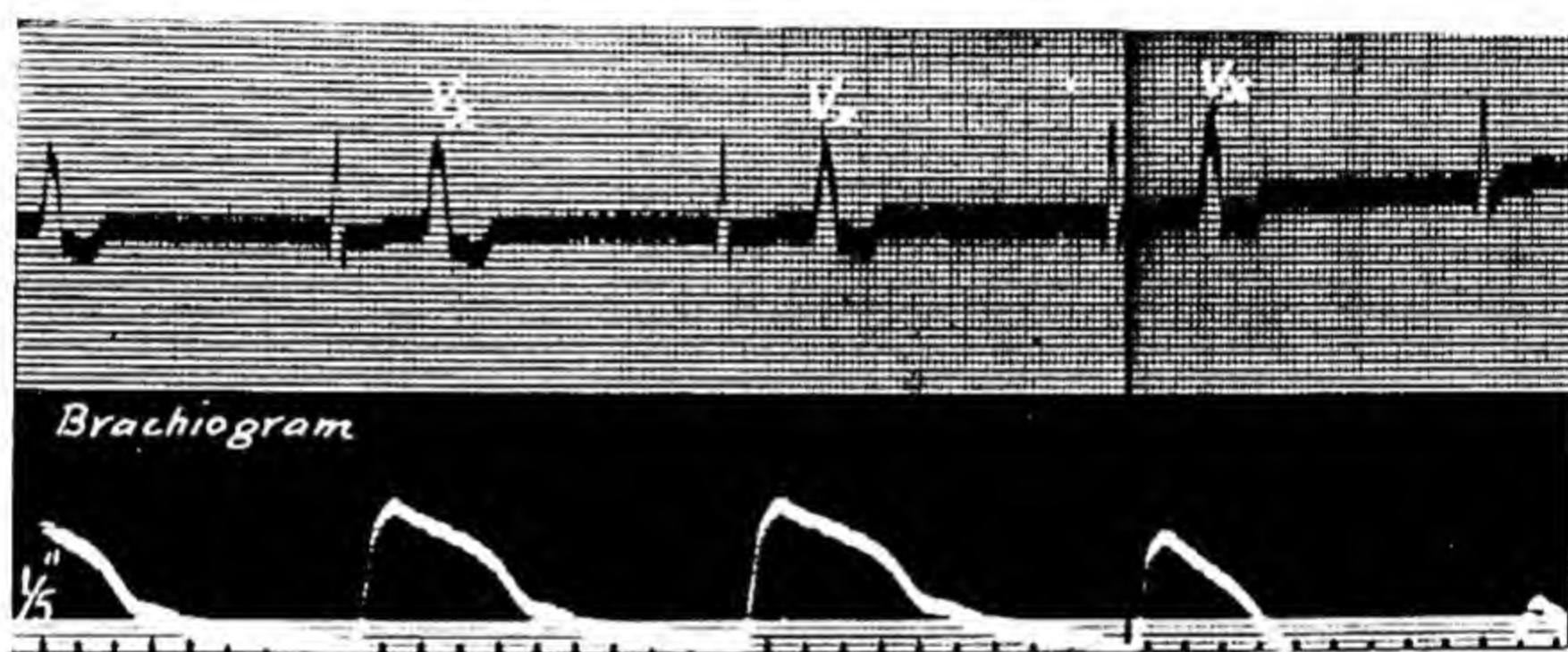


Fig. 52.—Premature Ventricular Beats, Bigeminy, Auricular Fibrillation. Every second beat is a ventricular extrasystole ( $V_x$ ). Auricular fibrillation is present but the normal ventricular beats come regularly, *i.e.*, complete heart block. Such curves result from toxic doses of digitalis. Lower tracing is from the brachial artery and shows that the extra beats do not reach the wrist. Apex rate 60, pulse rate 30. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

case the P-R interval may also be delayed (Fig. 53), or if auricular fibrillation was present (Fig. 52). When this occurs it indicates a toxic effect of digitalis and the drug must be omitted or the dose should be diminished. Coupling due to extrasystoles, however, may occur without digitalis and even in the absence of heart disease.

Ordinarily it is not difficult to recognize extrasystoles at the bedside. When they are numerous they may resemble auricular fibrillation but they may generally be differentiated by the method discussed in a previous paragraph. It is regarded as impossible to distinguish auricular from ventricular premature beats without graphic methods. One may obtain certain suspicions that help to identify the one from the other. If the observer taps with his foot in rhythm with the regular beats, and continues the same pace when the extrasystole occurs the foot will come down synchronously with the following beat if it is a ventricular extrasystole. This will generally not be so if it is auricular because the post-



extrasystolic pause is not completely compensatory. Furthermore, the character of the sounds is different in the two types. An auricular extrasystole sounds more like the normal beat, only coming prematurely. A

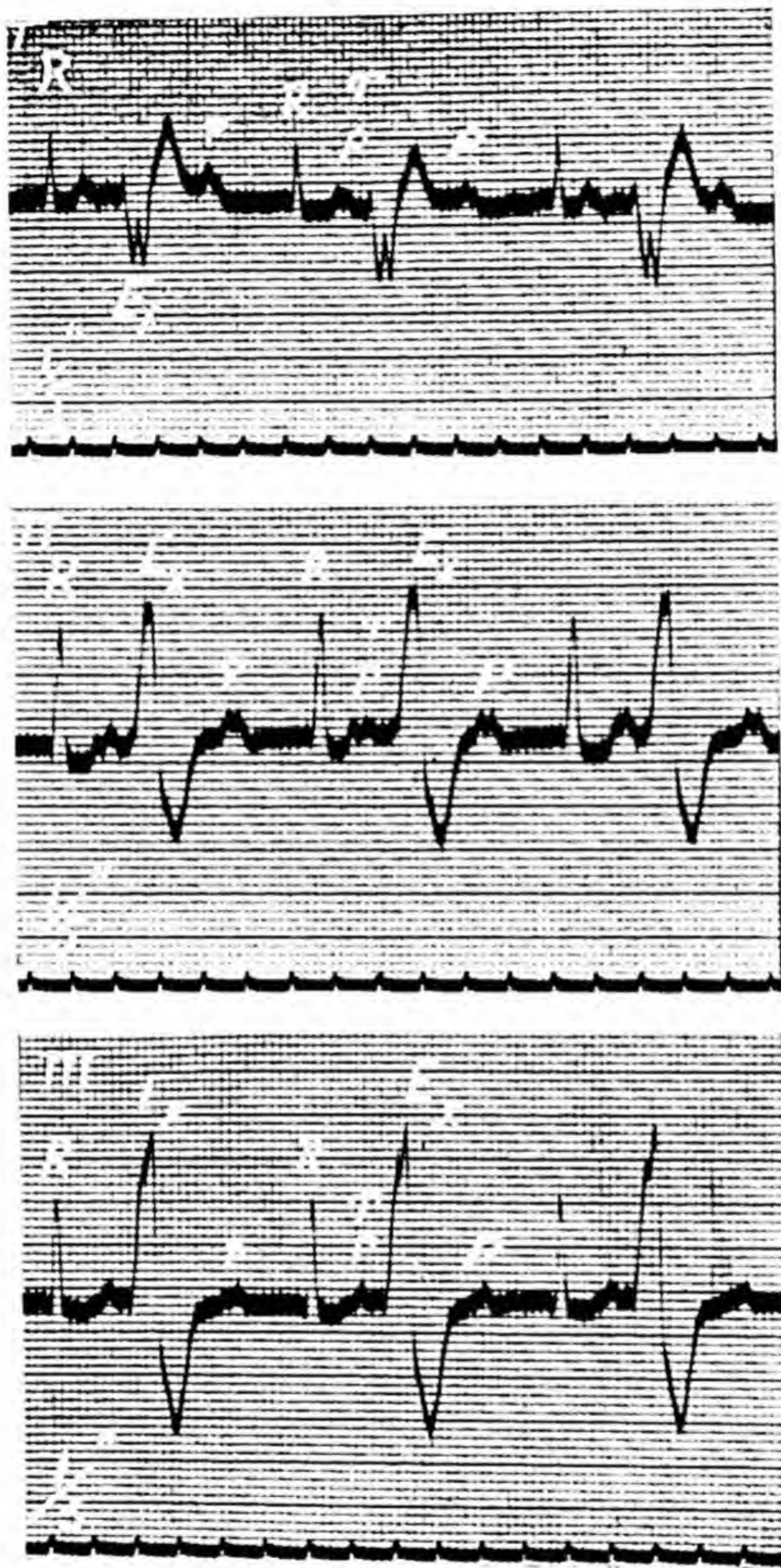


Fig. 59.—Digitalis Coupling. Every second beat is a ventricular extrasystole with the typical abnormal form. The a-v conduction time or P-R interval is markedly delayed, measuring 0.42 second (normal is 0.16 second). Both of these effects are typical of digitalis intoxication. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

ventricular extrasystole has a peculiar clicking sound which is different from the normal first heart sound of the particular patient, because the ventricles are apt to be contracting simultaneously with the auricles or at least in an abnormal relationship to auricular systole. This results in



another distinguishing sign that may be elicited. The fact that the auricles contract while the ventricles are in systole makes the auricular impulse in the jugular pulse more prominent and one may see a large "a" wave in the jugular vein. This takes place because the blood from the right auricle is prevented from going into the right ventricle and is

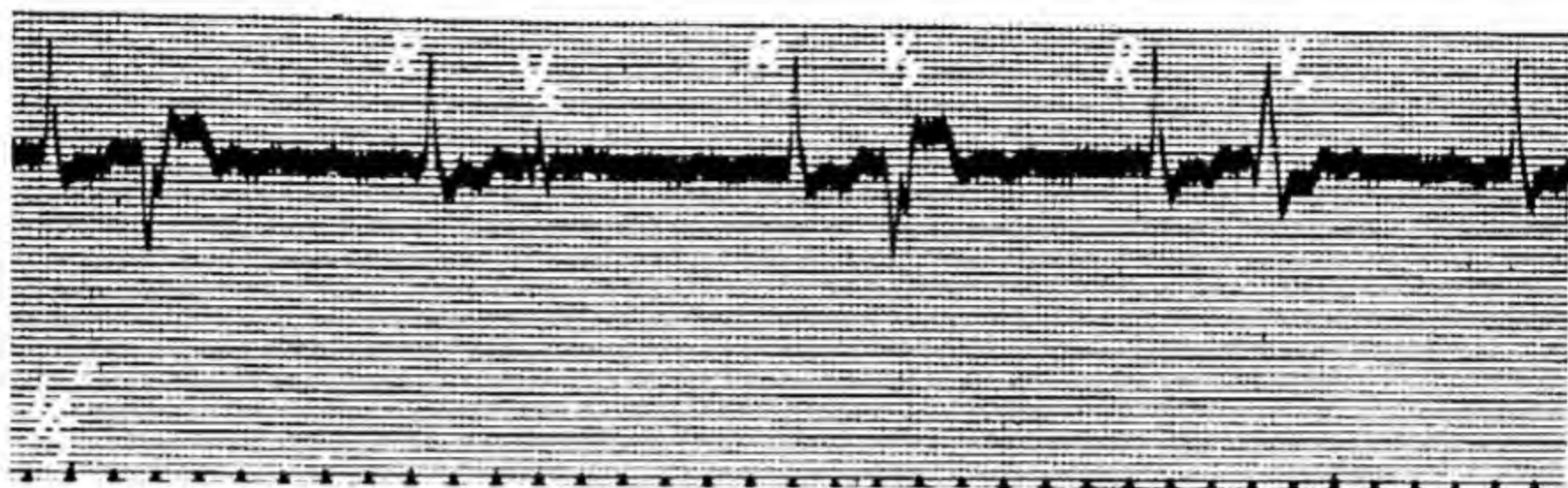


Fig. 54.—Multiple Premature Ventricular Beats. Following each normal beat (R) there are ventricular extrasystoles having varying forms ( $V_x$ ,  $V_y$ ,  $V_z$ ). These beats arise in different foci in the ventricles or have different conduction paths. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

forced back through the superior vena cava. However, when it is important to identify the type of extrasystole with certainty graphic methods will be necessary.

The *main symptom* that is produced by ventricular extrasystoles is palpitation. This will be variously described by different individuals and

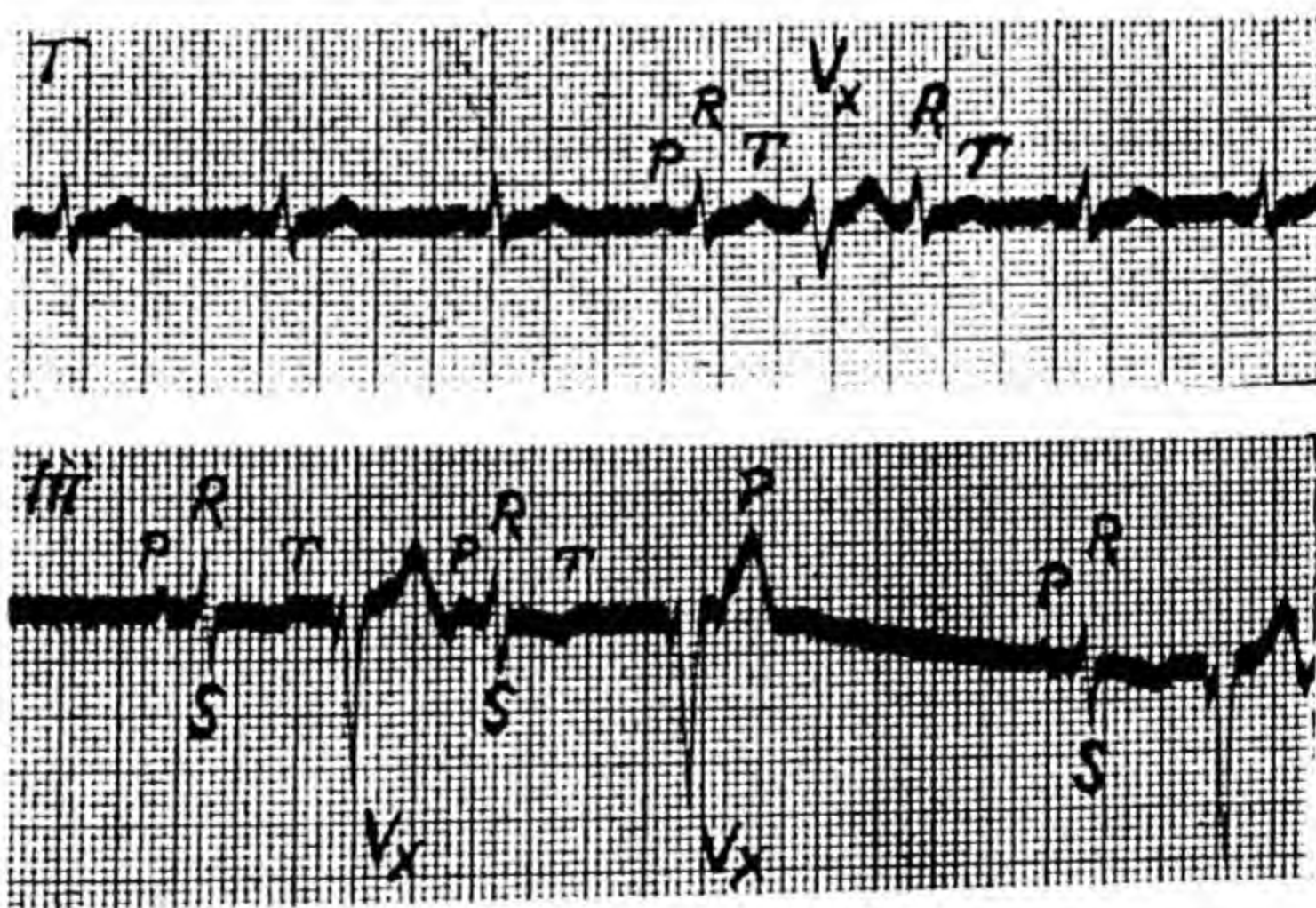


Fig. 55.—Interpolated Premature Ventricular Beats. Upper tracing shows a single ventricular extrasystole ( $V_x$ ) interpolated between two normal beats. Lower curve shows both an interpolated ventricular extrasystole and an extrasystole followed by the customary compensatory pause. The first patient had no heart disease, the second had angina pectoris.

generally the diagnosis can be made from the history itself. The various expressions used are "skipping of the heart," "a sudden flop or hesitation," "the heart flutters for an instant and then stops," "a thump in the neck or chest" (probably due to the beat after the pause which is always a vigorous contraction), "a sudden sinking or faint sensation or a wave,"



and other terms often very graphic in their description. Occasionally a momentary darting pain occurs with each extrasystole. In many individuals they produce no symptom and then are accidentally found on examination. This is more commonly the case in stout patients and in those who are more phlegmatic. It seems that with a thin chest the somatic disturbances within the chest cavity are more readily felt. They are most frequent and most troublesome at rest, especially while a patient is trying to fall asleep, and often are entirely absent during physical activity. This may be due to the fact that as the heart slows with rest, the diastolic pause lengthens and there is a greater opportunity for a premature beat to arise during longer than during shorter pauses. There are other neurogenic factors that determine the development of these beats at one time or another apart from the heart rate. There is no doubt that emotional disturbances can be responsible for their presence. The following experience is quite illustrative. This man who had the typical complaint of skipping of his heart did not happen to show any irregularity during my examination. The symptom first began after the death of his child nine months before. While his electrocardiogram was being taken and the heart was beating regularly I asked him what illness his child had, and as he began to speak about his child the extrasystoles appeared. This fits in very well with the physiological work showing that there is a center in the hypothalamic region which controls ventricular extrasystoles.

There is another type of ventricular extrasystole that only develops on physical effort. In general this is more serious and may even predispose to the development of ventricular tachycardia on effort. Occasionally in certain individuals they are produced by coffee or smoking. More often when this is thought to be cause and effect, carefully controlled observation will show that there is no such relationship. Often no known cause can be detected for their presence.

As to *treatment*, it is best for the physician to regard extrasystoles lightly. The patient should be clearly told that they have no serious significance and do not mean heart disease. Some persons have them all their lives and are not handicapped thereby. Such individuals should not be restricted in their activities unless there is some other reason for such restriction. When extrasystoles produce no symptoms no medication should be advised. When they are troublesome quinidine sulfate 0.2 to 0.3 gram (3 to 5 grains) two or three times a day or taken just before they usually occur may eliminate them entirely. Occasionally moderate doses of digitalis, curiously enough, also inhibit extrasystoles. Strychnine (0.001 gram— $\frac{1}{80}$  grain—three times a day) has also been recommended. In some cases I have found that the extrasystoles disappear when the patient is given 2.0 grams (30 grains) of potassium phosphate or chloride three times a day. Atropine sulfate may diminish vagal tone and increase the heart rate sufficiently to prevent their development. In general it may be said that when it is important to eliminate extra-



systoles, one or another of the various drugs that are available will prove effective in most cases.

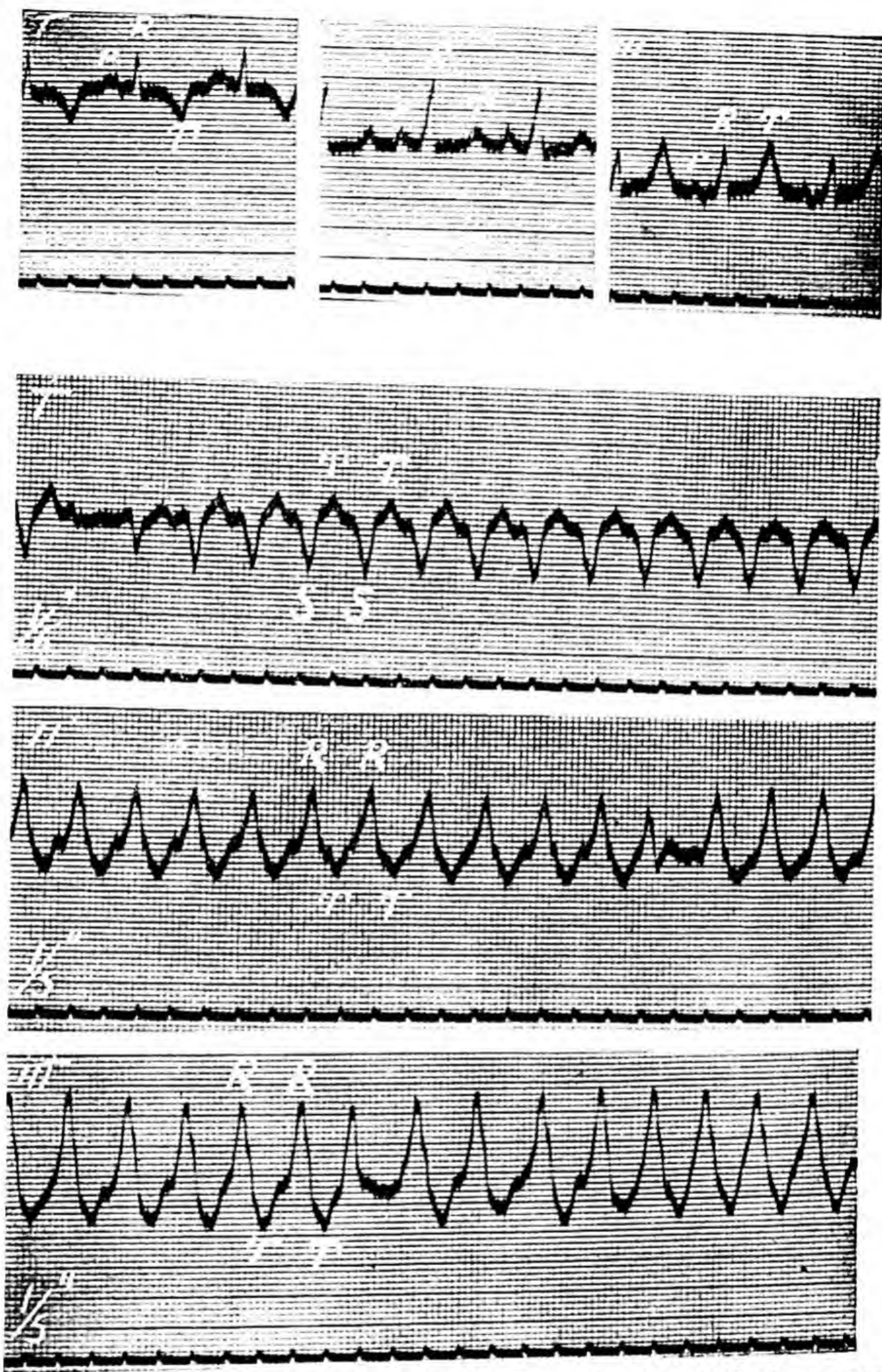


Fig. 56.—Paroxysmal Ventricular Tachycardia. The upper three leads show a normal rhythm, rate 89. The lower three leads were taken the next day during an attack of tachycardia, rate 172. Note that the form of the ventricular complexes (RST) changed markedly in the corresponding leads. There are slight irregularities in the length of the heart cycles during the tachycardia. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

**Paroxysmal Ventricular Tachycardia.**—An ectopic ventricular rhythm of still higher grade is paroxysmal ventricular tachycardia. This may



be regarded as a consecutive series of ventricular extrasystoles arising from an ectopic focus in the ventricle. There is much in the nature of this mechanism that resembles a circus motion and it may eventually be proved to be a disturbance in the ventricles similar to flutter in the auricles. Starting as it does from an abnormal focus in the ventricles, the impulse travels an abnormal course and the resultant ventricular complex will be abnormal (Figs. 56 to 58). Each individual complex resembles a ventricular extrasystole. The impulse may travel in a retrograde fashion up the junctional tissue and produce auricular contractions. Because the rate is rapid, there may be a retrograde block so that only every other ventricular impulse reaches the auricles. At other times the

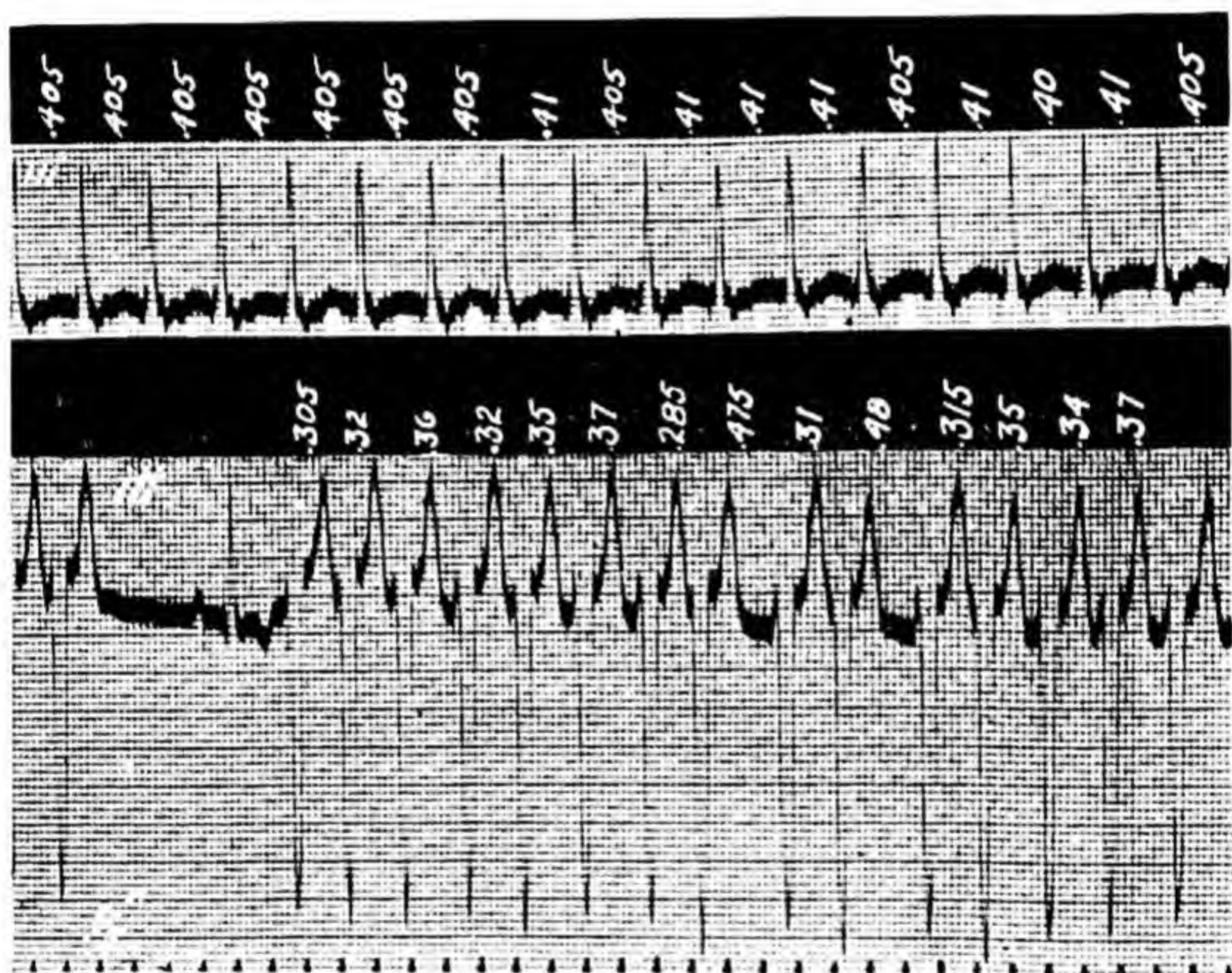


Fig. 57.—Paroxysmal Ventricular Tachycardia. The upper tracing was made during an attack of auricular tachycardia; the lower one shows the onset of ventricular tachycardia, occurring in the same patient. The numbers indicate the length of the heart cycles. Note the absolute regularity of the auricular tachycardia and the distinct irregularity of the ventricular tachycardia.

auricles contract independently and follow their own pacemaker in the sino-auricular node. It is often impossible to identify the P waves because they are buried in the QRS-T complexes.

It may be difficult to distinguish ventricular tachycardia from a rapid auricular rate with a block in one or the other bundle of His. Both conditions will show similar ventricular complexes. The finding of isolated ventricular extrasystoles while the heart is beating slowly which resemble the complexes when the rate is rapid or the identification of P waves that are not in the normal relationship to the ventricular complexes will prove that the condition is ventricular tachycardia.

For some hours or days after any attack of paroxysmal rapid heart



action the ventricular complexes may remain distinctly abnormal showing inverted T waves. These changes may occasionally occur when the heart is structurally normal and therefore cannot necessarily serve as evidence of heart disease. They probably indicate heart muscle fatigue or local relative anoxemia of parts of the ventricles but the curves return to normal eventually. Lack of appreciation of this will lead to incorrect diagnoses of coronary or myocardial disease.

There are numerous important clinical aspects of paroxysmal ventricular tachycardia and this disturbance needs to be differentiated from other forms of rapid heart action. It can occur as paroxysms or it may remain permanent if treatment is not instituted. The rate is apt to be around 160 to 180 and very rarely reaches the high levels of 220 or more that occur in auricular tachycardia. Unlike the latter it generally accompanies grave heart disease, especially disease of the coronary arteries, but it may be present as a purely functional arrhythmia in an otherwise healthy heart. Furthermore, there are bedside

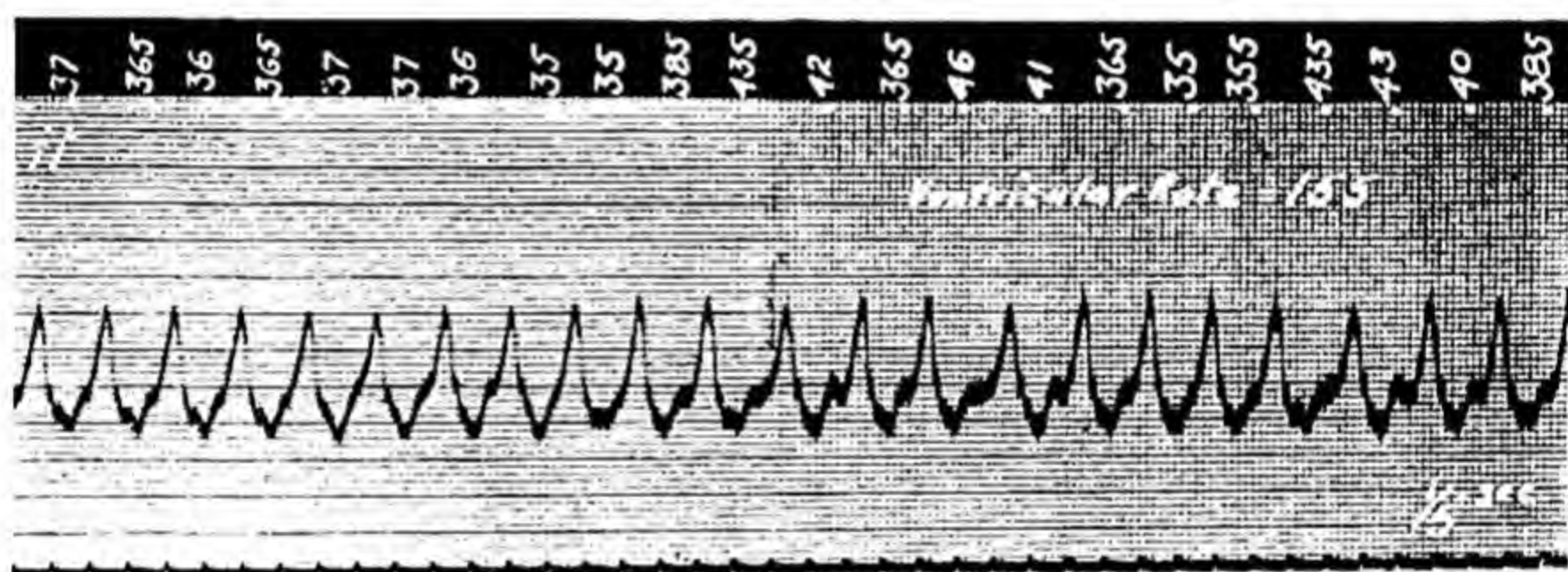


Fig. 58.—Paroxysmal Ventricular Tachycardia. Numbers above indicate the interventricular intervals in hundredths of a second. Note that although the first nine cycles are quite regular, distinct variations in rate occur thereafter.

methods that enable one to diagnose this condition. When paroxysmal, the attacks begin and end suddenly (Fig. 57). The rate is rapid but in most cases slight irregularities can be detected on auscultation in marked contrast to auricular tachycardia (Fig. 57). Occasionally the rhythm will be perfectly regular but more often, if one listens long enough, interruptions will be heard. The first nine cycles in Figure 58 are just as regular as the cycles that occur in auricular tachycardia but thereafter appreciable differences in the length of the cycles develop that will never be seen in association with the latter condition. Furthermore, on auscultation slight but definite differences in the intensity and quality of the first heart sound will be heard, which are due to the different relationship between the ventricular and auricular systoles in various cycles. This latter auscultatory finding will not be present either in normal tachycardia or in paroxysmal tachycardia. It also has been pointed out that the auricular pulsations as seen in the jugular vein will be fewer in number than the ventricular rate, and some of these jugular pulsations are particularly conspicuous. Finally this type of rapid heart action is never



influenced by any of the methods used to stimulate the vagus such as carotid pressure, ocular pressure or deep breathing.

Therapeutically the problem of ventricular tachycardia is peculiar. In most cardiac conditions in which there is heart failure digitalis is indicated and a beneficial, or at least not harmful, effect is anticipated. When this arrhythmia is present, digitalis will not only fail to improve the situation but may well worsen it. I have seen two instances in which digitalis, on repeated trials, accelerated the ventricular rate while the tachycardia was in progress and aggravated the state of the circulation. Quinidine, on the other hand, is almost a specific drug for this type of tachycardia. In most cases it will restore the normal rhythm. The amount

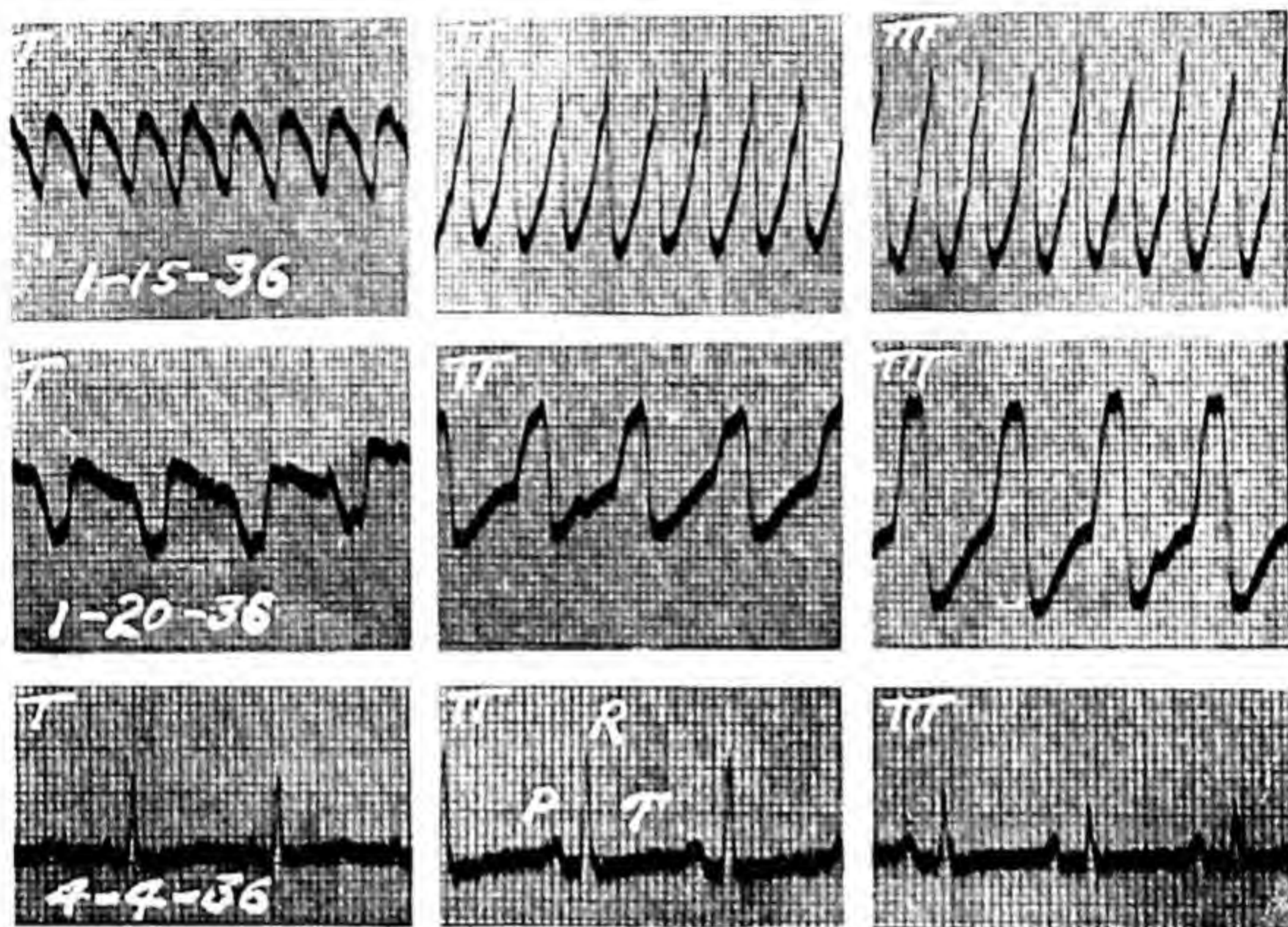


Fig. 59.—Paroxysmal Ventricular Tachycardia—Quinidine Effect. First set shows ventricular tachycardia, the rate is 247. Second set shows marked slowing of the rate (122) as a result of quinidine but ventricular tachycardia persists. Lowest tracing shows normal rhythm. Enormous amounts of quinidine were necessary to produce regularization, single doses being gradually increased from 0.2 gram to 2.0 grams. The patient was desperately ill with advanced congestive failure although after recovery there was no evidence of heart disease.

necessary to accomplish this will vary greatly from a single dose of 0.8 gram (5 grains) to as much as 1.5 grams (22 grains) administered five times a day. Before the heart becomes regularized the ventricular rate will gradually slow (Fig. 59). This slowing enables one to follow the effect of the drug and guide its dosage. On rare occasions even large doses of quinidine will only slow the ventricular rate but fail to do away with the abnormal mechanism, the original rapid rate returning as the effect of the drug wears off. On two such occasions 2 milligrams ( $\frac{1}{80}$  grain) of atropine sulfate, given subcutaneously about one hour after a large oral dose of quinidine, promptly restored the normal rhythm. The pharmacological action of atropine is to lengthen the refractory period



of cardiac muscle. Finally, 15 cc. of 20 per cent solution of magnesium sulfate intravenously has been used successfully.

**Ventricular Flutter?**—If one continues the sequence of abnormalities of ventricular origin, as was done in the auricles, one should naturally consider ventricular flutter. This generally has been omitted, as no con-

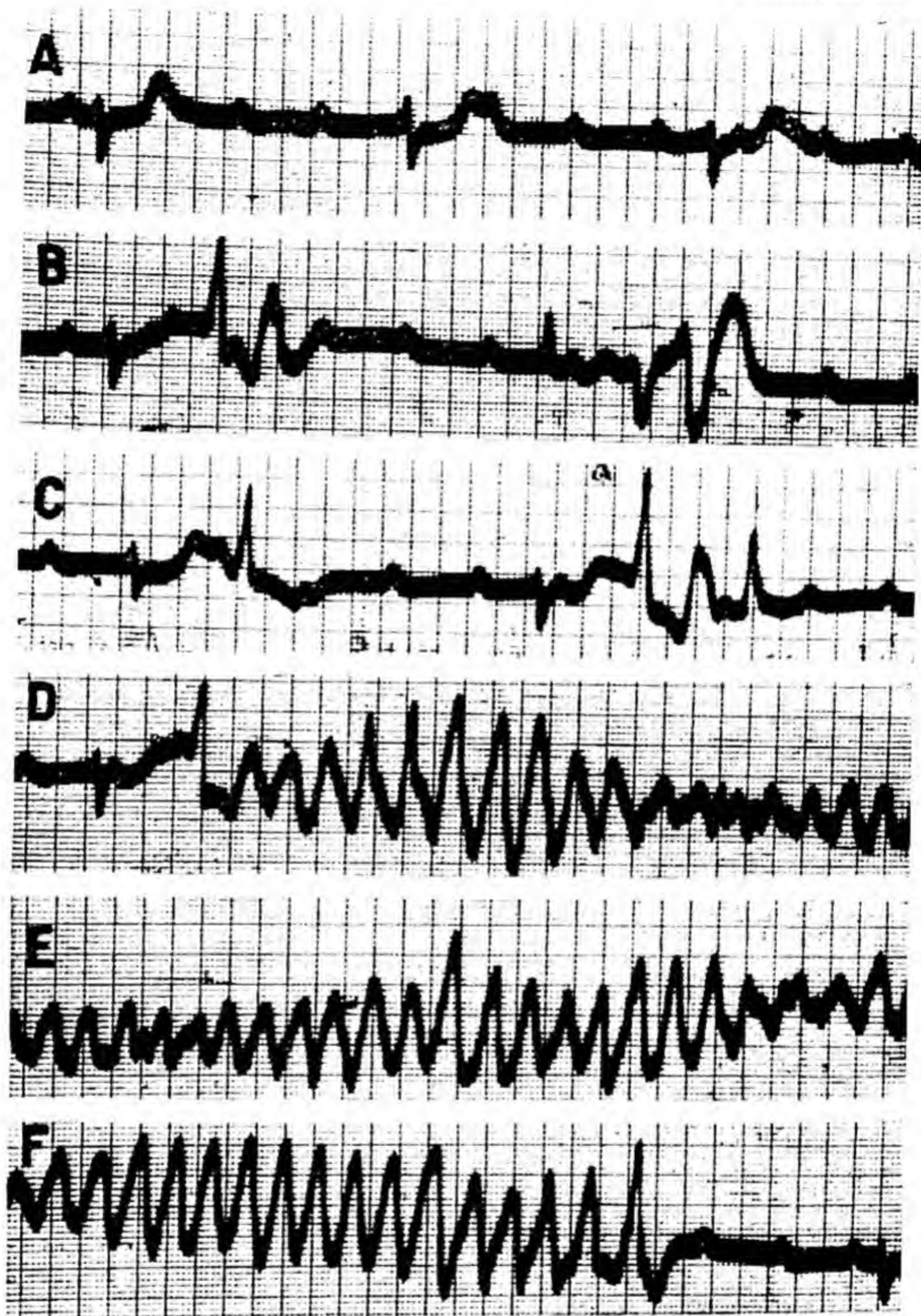


Fig. 60.—Ventricular Flutter (Possible). Strips of a continuous tracing (Lead I) from a woman fifty-nine years old with complete heart block, right bundle branch block and Adams-Stokes disease. A indicates complete block. B and C show ventricular extrasystoles of different types. D, E and F show oscillations that have been interpreted as ventricular fibrillation, but because heart beats were audible throughout this period it is suggested that it be called ventricular flutter. The last complex shows return of complete block.

dition has been recognized as ventricular flutter in the electrocardiograms. Figure 60, however, may possibly represent such a condition. These rapid, slightly irregular oscillations were taking place at the very time that very rapid heart beats could be heard. The patient was unconscious. However, she recovered from this attack. Ordinarily one has



considered such curves as signifying ventricular fibrillation, but the presence of heart beats and the fact that the circulation was being maintained militate against this diagnosis and denote that the ventricles were actually contracting.

**Ventricular Fibrillation.**—The final and most extreme disturbance of the ventricles is ventricular fibrillation. With this there are numerous impulses traversing the ventricles so rapidly that coordinated contrac-

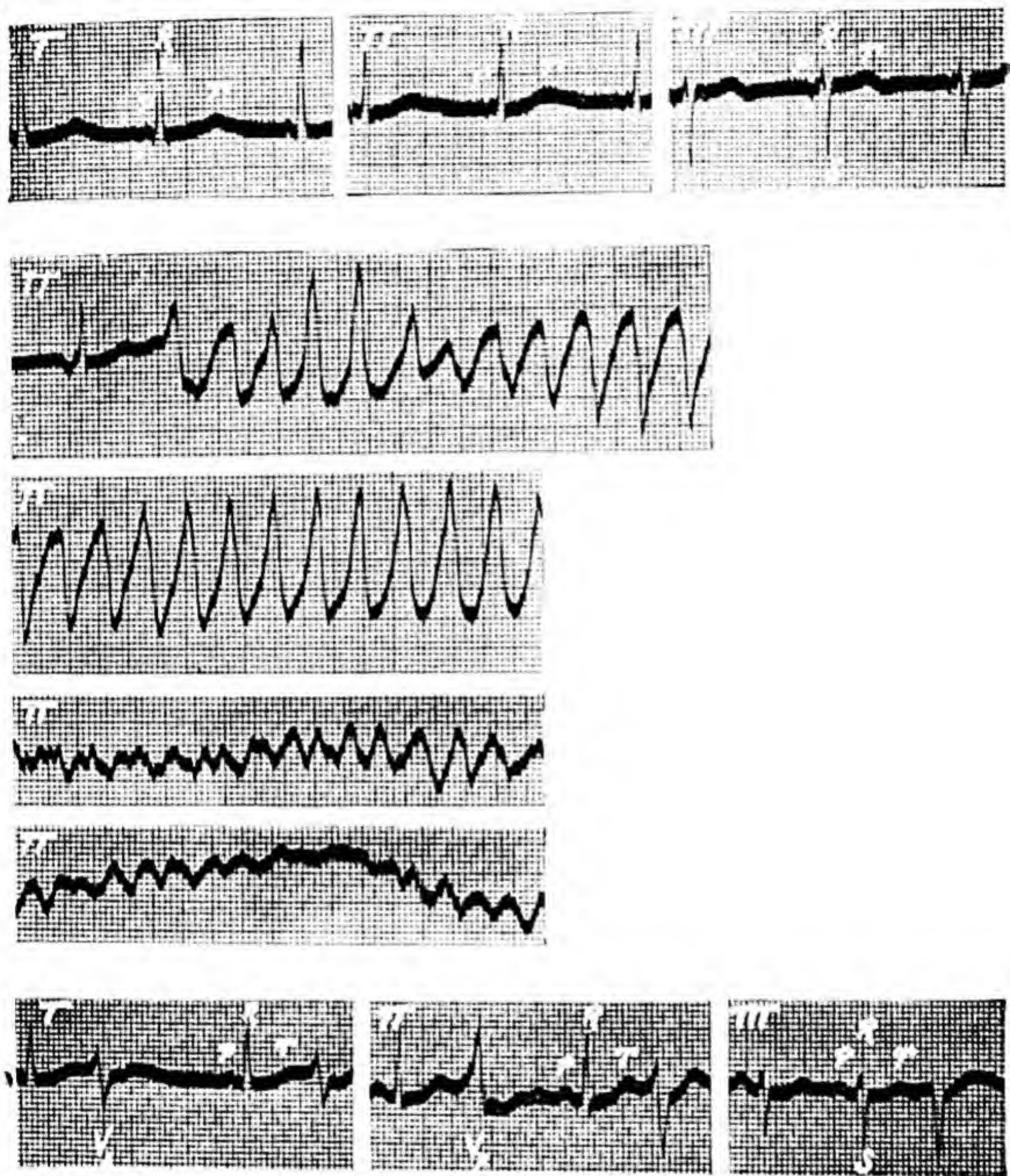


Fig. 61.—Ventricular Fibrillation. The upper three leads show a normal mechanism. The following four curves are portions of a continuous tracing taken during an attack of syncope and are characteristic of ventricular fibrillation. The patient had a convulsion and there was no heart beat or pulse for about one minute. The lowest three leads show a return to a normal beat with occasional ventricular extrasystoles ( $V_x$ ). The patient became ambulatory.

tions do not occur. When the condition is reproduced experimentally by faradizing the ventricles, tying the branches of the coronary arteries or by drugs such as digitalis and adrenalin, fibrillary twitchings will be seen but no mechanical expulsion of blood results. From the point of view of the dynamics of the circulation the heart suddenly stops. The electrocardiograms that represent this state show very bizarre, rapid and irregular ventricular complexes (Figs. 61, 62). The initial phase of



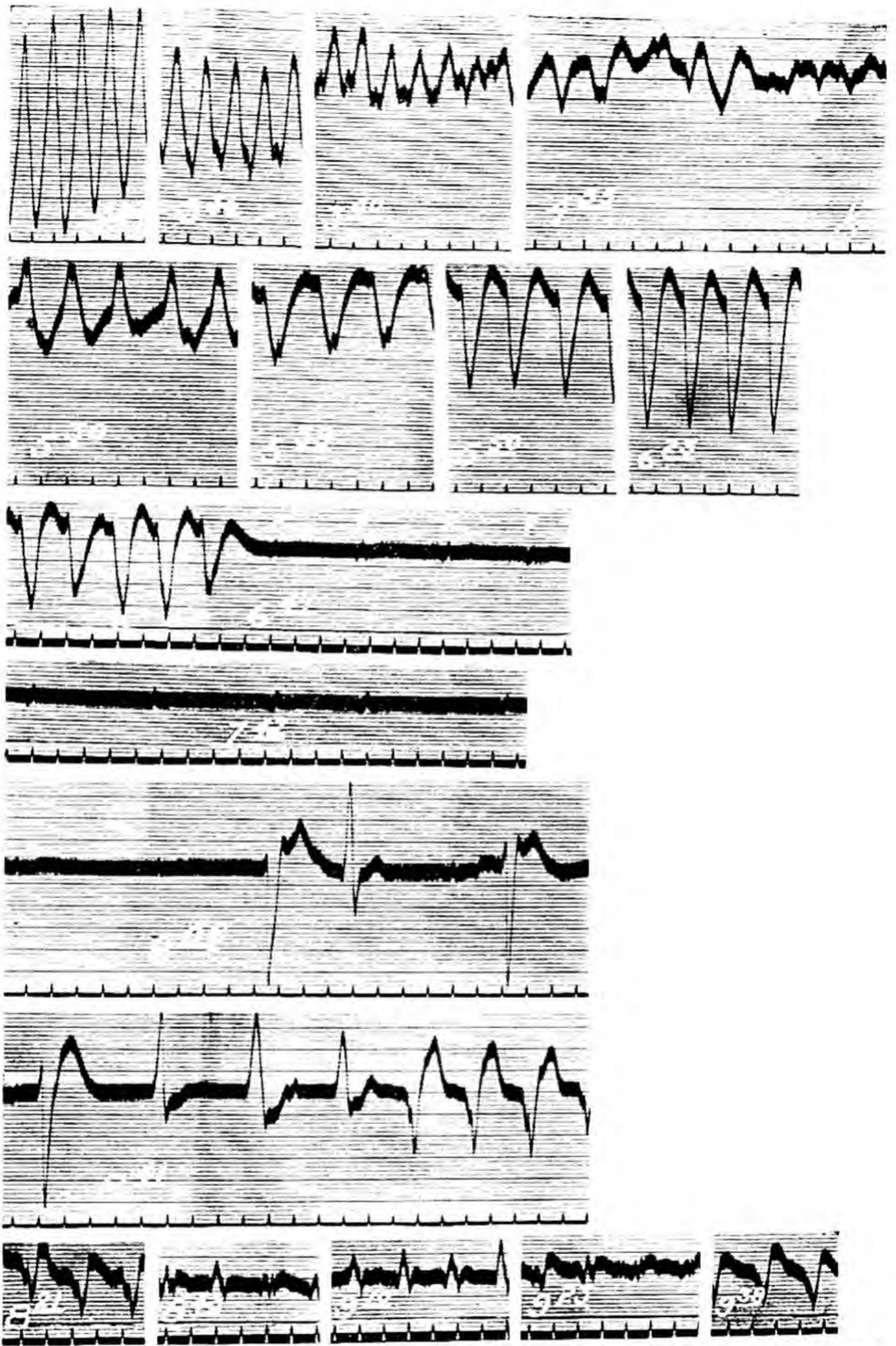


Fig. 62.—Ventricular Fibrillation. Portions of a continuous tracing taken during an attack of Adams-Stokes syncope lasting five minutes. The numbers indicate minutes and seconds. The patient was unconscious throughout the attack, breathing ceased and there was no detectable heart beat. Note the early development of ventricular fibrillation with eventual cessation of all electrical activity except for small auricular waves (P). During this time adrenalin was injected directly into the heart and contractions were resumed. The patient recovered and became ambulatory. (Published in "Heart," vol. 12.)

the ventricular waves (QRS) is broadened and the T waves fuse with them so that they become indistinguishable. They also vary in height



from oscillation of large amplitude to coarse low movements of the base line.

It is extremely rare to find ventricular fibrillation in clinical practice because in most cases it is the cause of instant death. By mere chance, examples have been recorded when a patient happened to have an attack while the electrocardiograms were being taken. This was the case in an instance of sudden death from an attack of angina pectoris in which the postmortem examination showed disease of the coronary arteries but no acute thrombosis. This mechanism probably is a common cause of instant death in disease of the coronary arteries and from such causes as electrocution. Figure 63 shows the recording made when a patient happened to have an attack while electrocardiograms were being made. This patient had previous angina and had had a recent acute myocardial infarct. He was doing quite well and just after the first lead of the elec-

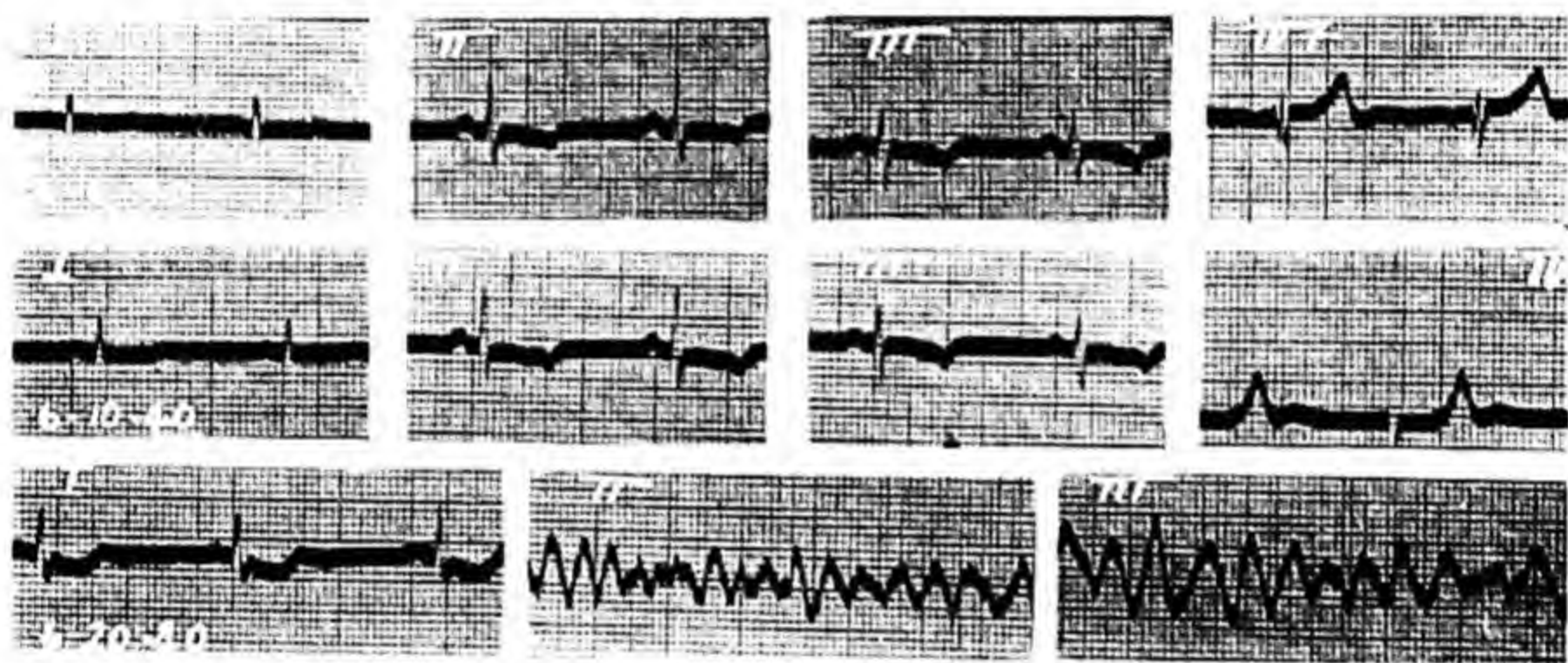


Fig. 63.—Ventricular Fibrillation (Cause of Sudden Death in Coronary Disease). This patient, a man sixty years old, had typical angina for three weeks with many attacks daily, promptly relieved by nitroglycerin. On the last day he had a severe attack requiring two injections of morphia and died instantly two hours after the onset of the last spell while tracings were being taken. Note the normal rhythm in Lead I of lowest set and ventricular fibrillation in Leads II and III taken only a few seconds later.

trocardiogram was taken he expired. Leads II and III, taken only a few seconds afterwards, showed that ventricular fibrillation was present.

Occasionally recovery takes place in human beings after ventricular fibrillation has set in. Figure 61 shows an example of this sort, in which repeated attacks of syncope were due to this mechanism and were generally preceded by brief periods of increased numbers of ventricular extrasystoles and ventricular tachycardia. The number of attacks seemed to be lessened by constant quinidine administration. In Figure 62 are shown not only periods of ventricular fibrillation but also complete cessation of all electrical responses in the ventricles, extending over a period of more than five minutes, with recovery. Whether the adrenalin that was injected directly into the heart was responsible for the recovery is not certain. This occurred in a woman about fifty years of age who was suffering from Adams-Stokes disease with attacks of unconsciousness.



## DISTURBANCES IN CONDUCTION

**Sino-auricular Block.**—Having considered the disturbances that may arise in the normal pacemaker of the heart and then the abnormalities in the origin of impulses (ectopic rhythms), the third main problem is to discuss the abnormalities in the conduction of impulses. The impulse after it is formed in the normal sino-auricular node may be blocked before it reaches the auricular musculature. This is called *sino-auricular block* (Figs. 64, 65). Neither the auricles nor ventricles receive an impulse and there results the loss of a complete heart cycle. If only one of those normal impulses is blocked, the pause will be approximately equal to two normal heart beats as the pacemaker remains undisturbed. Occasionally this pause will be equal to three or four heart beats in which case

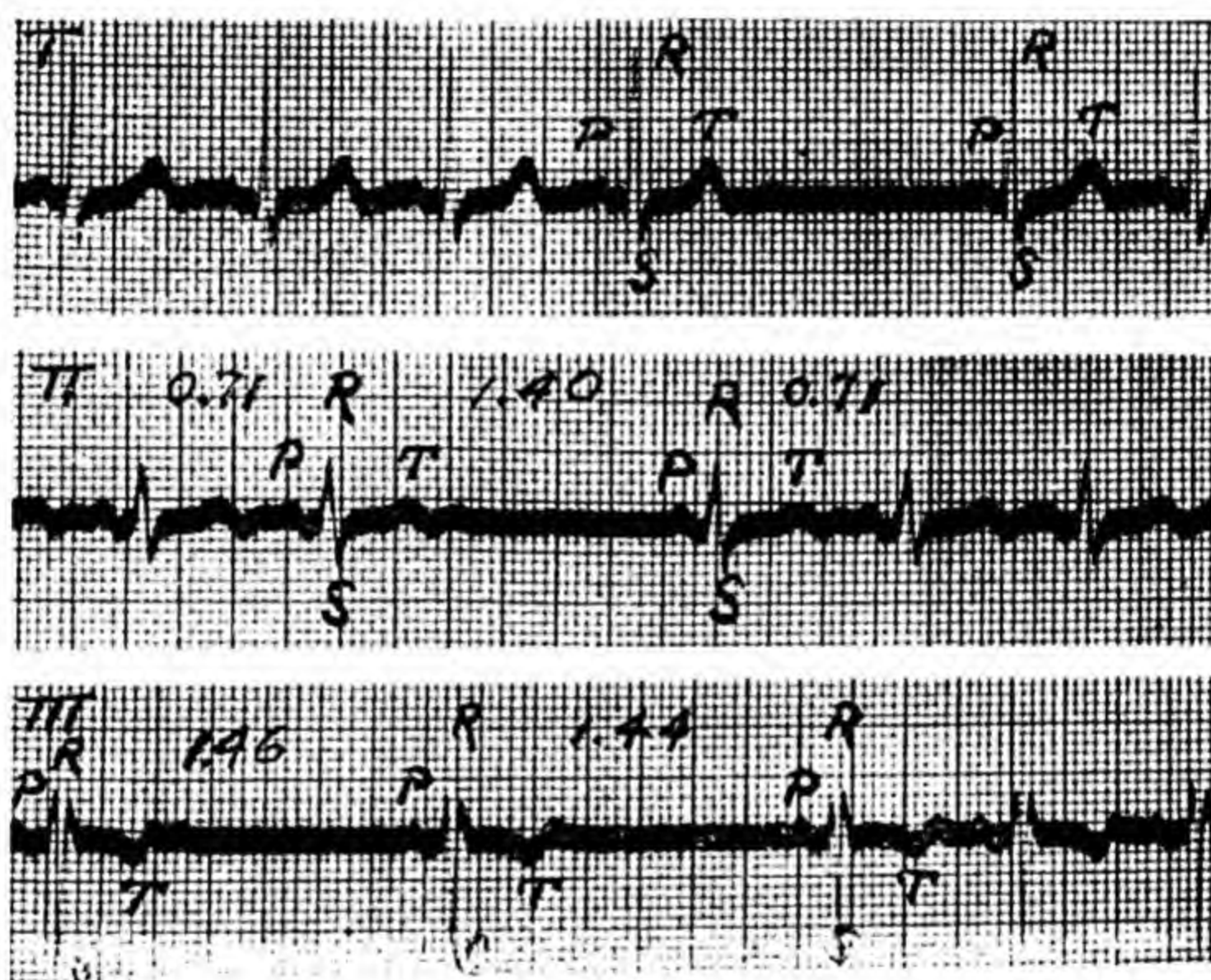


Fig. 64.—Sino-auricular Block. Note that the pauses (1.4 seconds) are equal to twice the length of the normal beat (0.71 second). Neither auricles nor ventricles contract during the pauses as the impulse is blocked at the pacemaker. The patient was a young woman with palpitation but without organic heart disease.

two or three impulses are blocked. Slight differences in measurements will be found because there often is an accompanying sinus arrhythmia that slightly affects the pace. Furthermore, after the pause the first beat may be an idioventricular beat or nodal escape.

Sino-auricular block is rare compared to auriculoventricular block. In general it occurs under the same conditions but has much less practical importance. It can be suspected clinically by detecting a complete loss of a heart cycle on auscultation. Because neither auricles nor ventricles contract during this pause, no auricular or "a" wave will be seen in the jugular pulse. If such a small wave is detected on careful examination of the veins of the neck during the long silent interval, the block is at the a-v node rather than at the s-a node. Treatment is generally not indicated for patients with this arrhythmia unless it produces syncope



when ephedrine or atropine may prove effective as in the ordinary type of block.

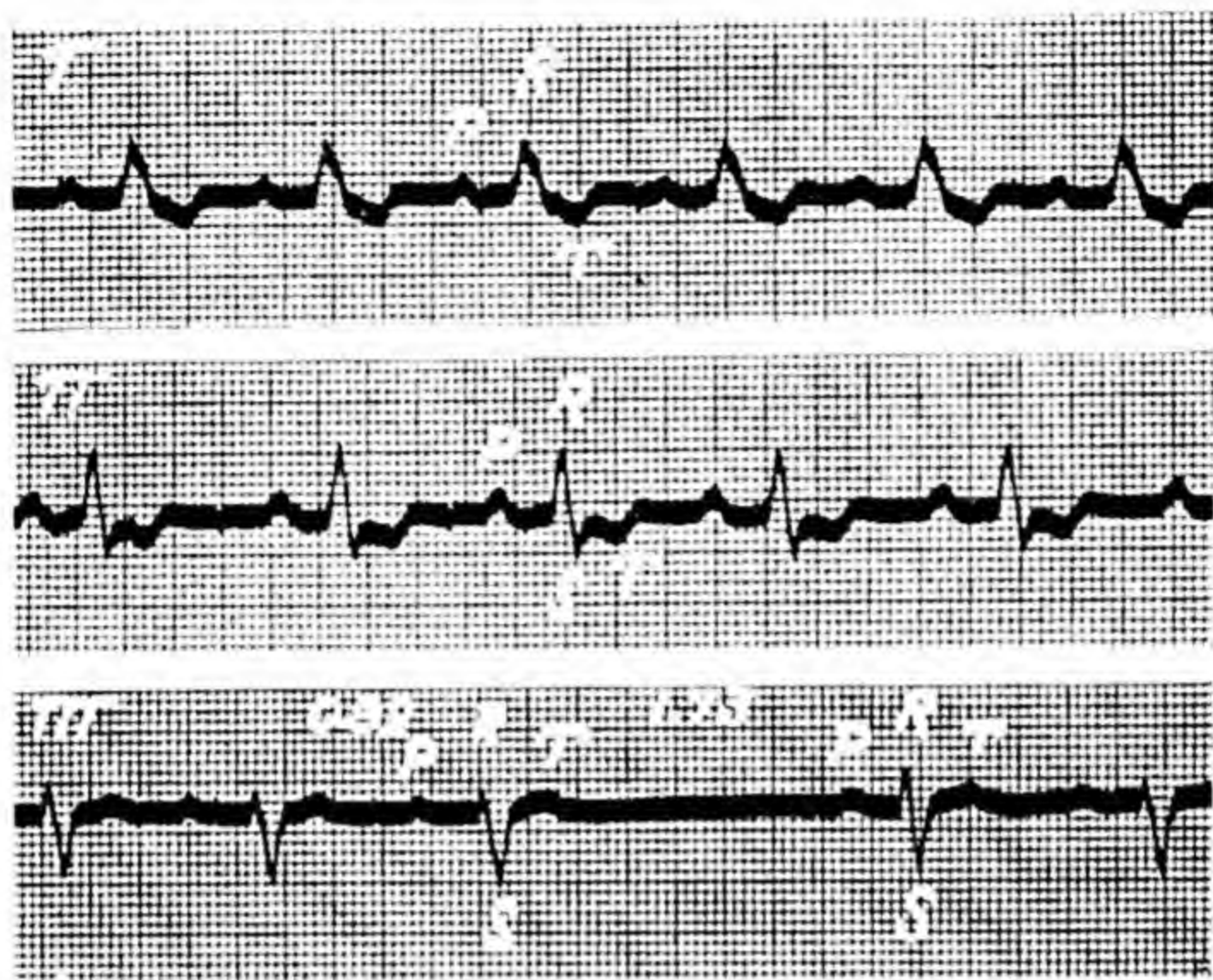


Fig. 65.—Sino-auricular Block. There is a pause in Lead III (1.73 seconds) approximately twice the normal heart cycle (0.92 second). Both auricles and ventricles failed to contract. Curves also show bundle branch block and delayed conduction time ( $P-R = 0.27$ ). The patient had hypertensive heart disease with coronary artery sclerosis.

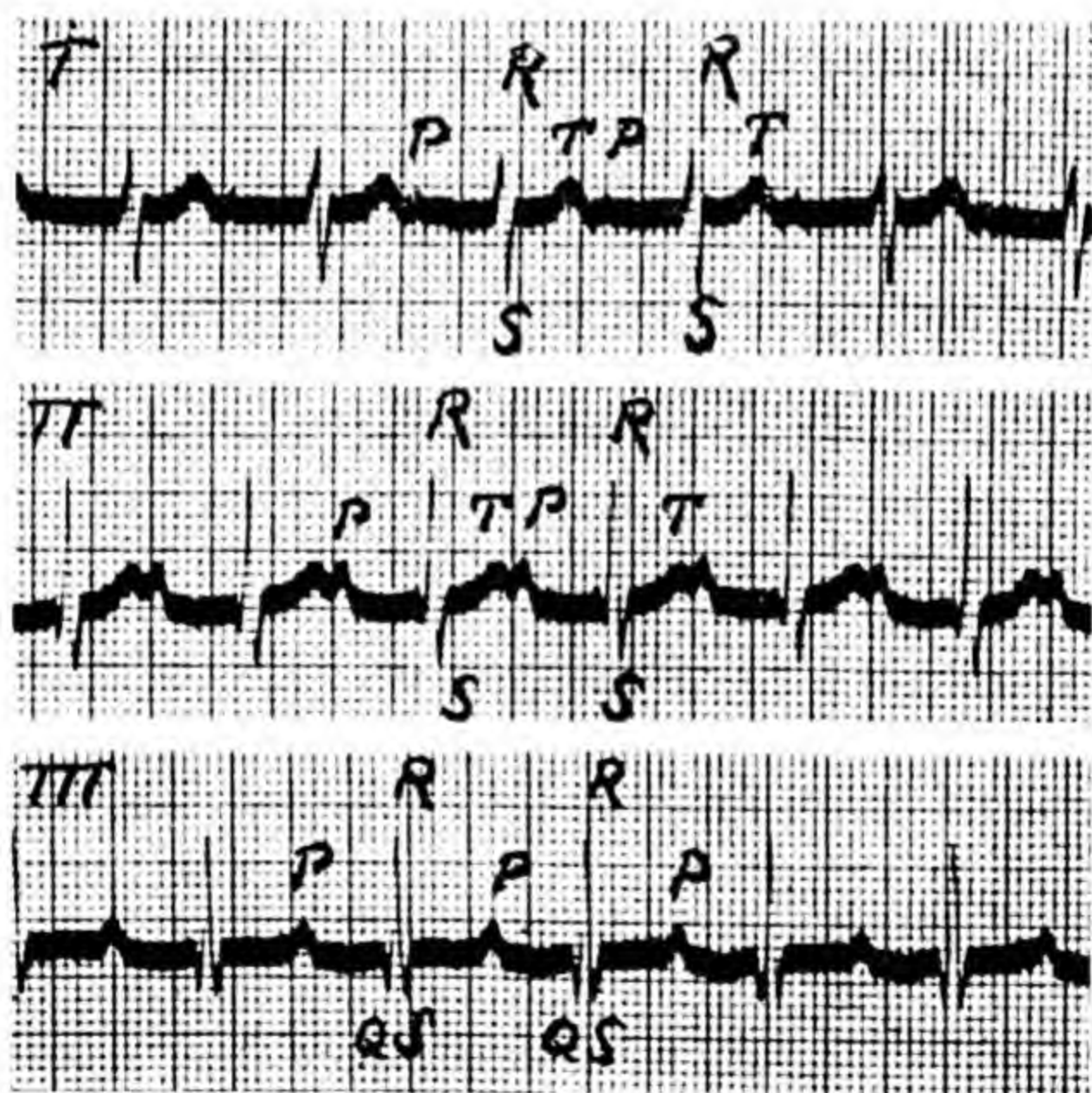


Fig. 66.—Heart Block, First Degree (Delayed Conduction Time). The P-R interval measures the time it takes for an impulse to go from auricles to ventricles and normally should be between 0.12 and 0.2 second. Here it measures 0.3 second. None of the beats are blocked so that the heart remains regular. The patient was suffering from postscarlatinal rheumatism.

**Auriculoventricular Block.—First Degree Heart Block (Delayed Conduction Time).—**The more common type of heart block is that which occurs in the junctional tissue, at the a-v node or bundle of His. When



the defect is slight, impulses are merely delayed in their passage through the main conduction path but eventually reach the ventricles. There

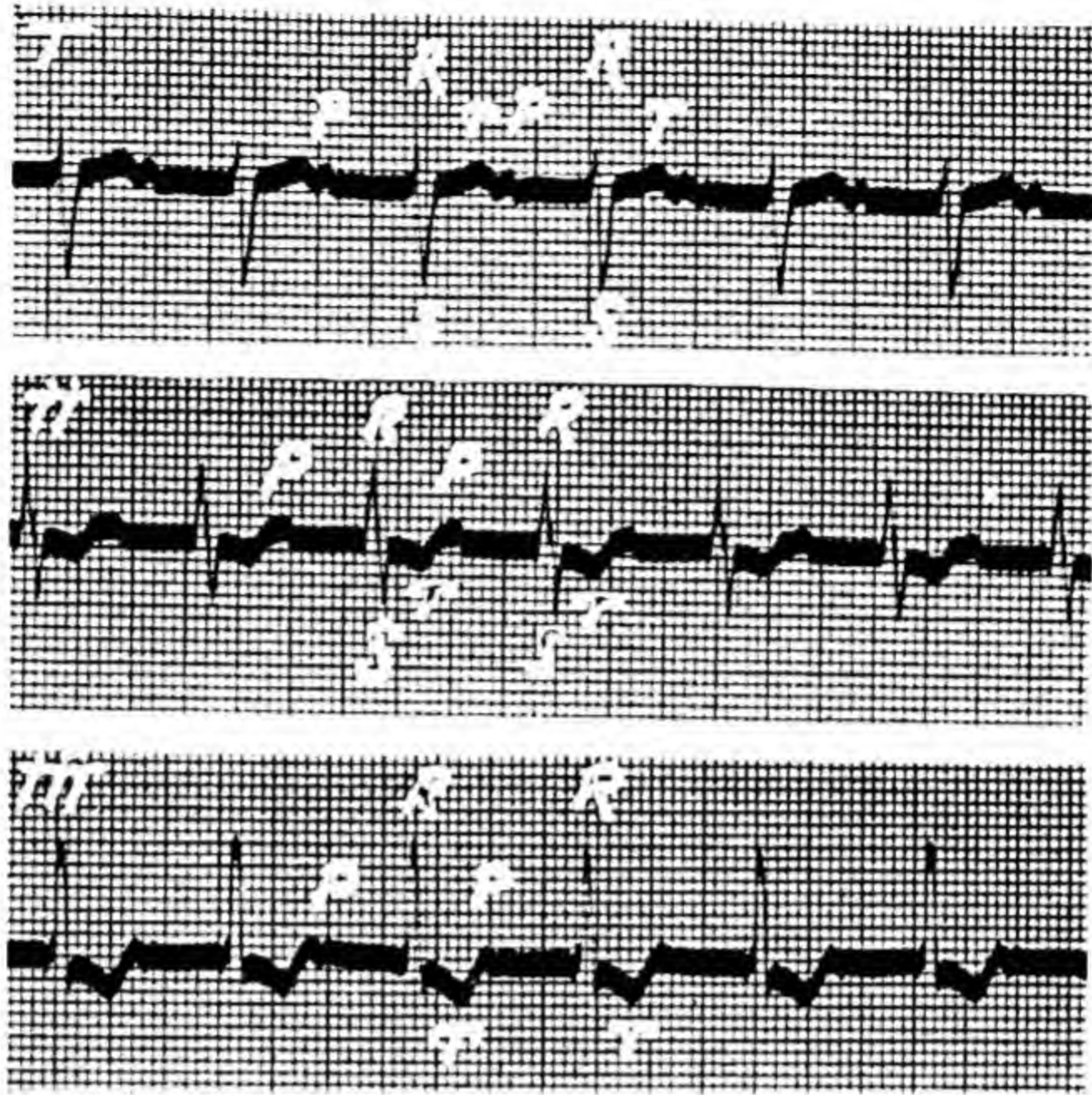


Fig. 67.—Heart Block, First Degree. The P-R interval is markedly delayed, measuring 0.32 second, but no beats are blocked. The patient had rheumatic mitral stenosis and insufficiency.

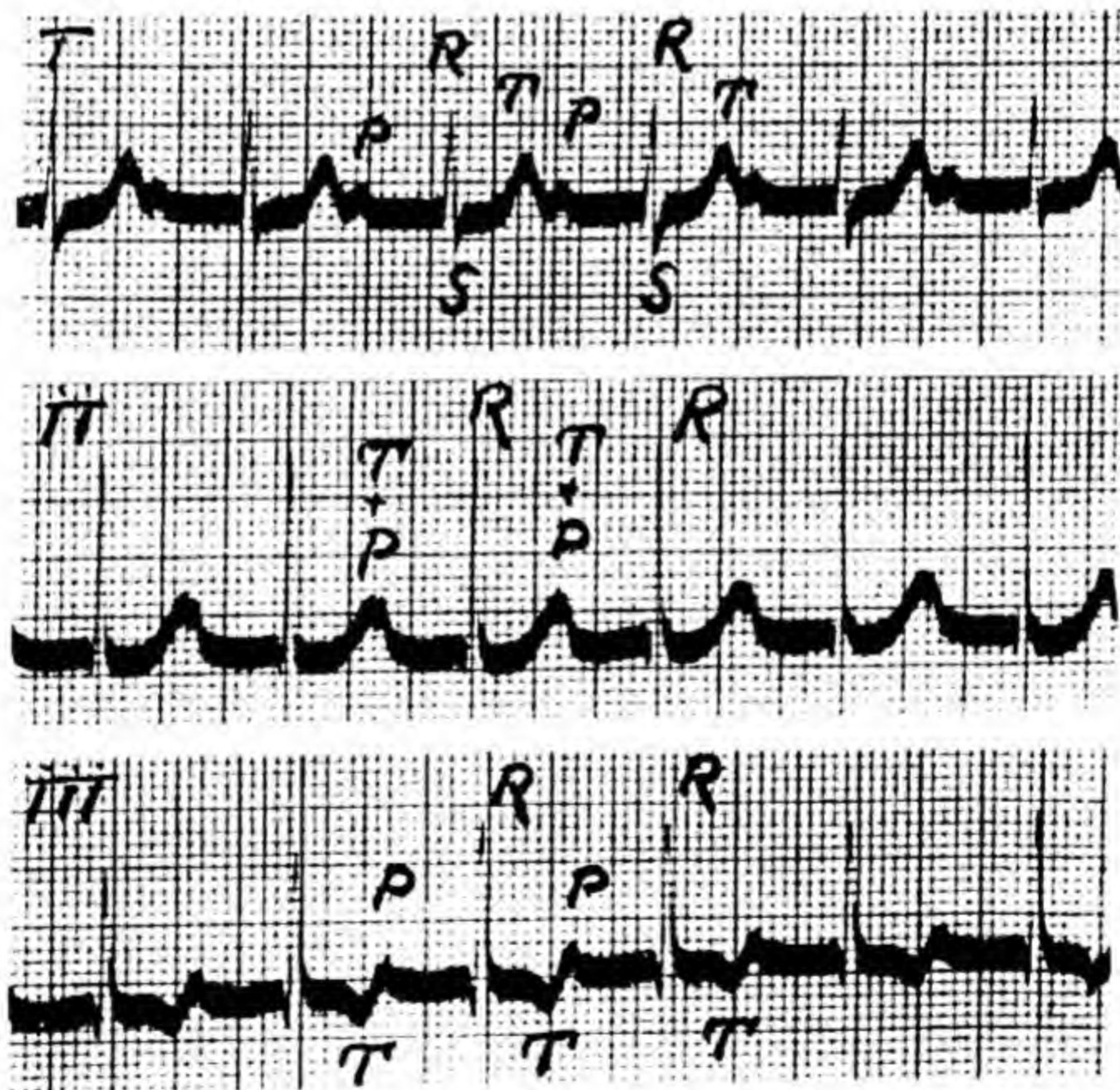


Fig. 68.—Heart Block, First Degree. The P-R interval is delayed to 0.32 second but the rhythm remains regular. Note that in Lead II the P wave is practically fused with the preceding T wave. The patient was a boy of thirteen who had an apparent "cold," the electrocardiographic changes being the only evidence at that time that the infection was rheumatic.

really is no blocking of beats and the heart remains regular. The conduction time (P-R interval) which normally measures about 0.16 second



becomes prolonged and when it reaches the duration of more than 0.2 second it is arbitrarily regarded as an indication of pathologic change (Figs. 66 to 70). The actual path that the impulse travels is normal and therefore the electrical complexes are all normal in form. The P wave

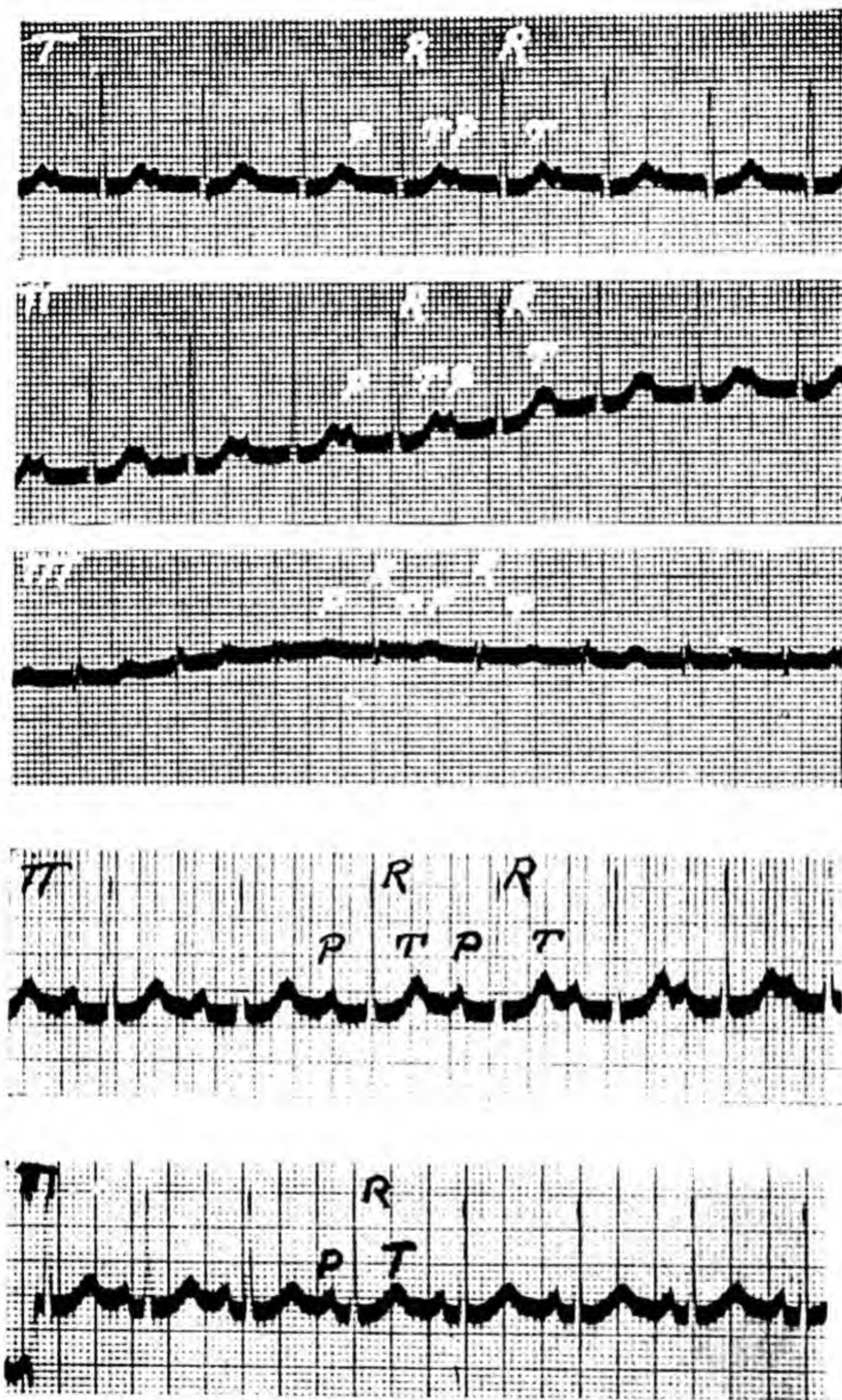


Fig. 69.—Heart Block, First Degree. The upper three leads, taken June 20, 1935, show delayed P-R interval of 0.9 second. The middle curve, taken July 5, 1935, shows that the P-R time is much shortened, 0.23 second. The lower set, taken September 5, 1935, shows normal P-R interval (0.16 second). The patient was a girl of thirteen with vague abdominal symptoms, who was sent to the surgical service for appendectomy. The electrocardiograms were the main clue that she had rheumatic fever.

may precede the ventricular complexes sufficiently to fall on the previous T or R wave. Under such circumstances it may become difficult to recognize the P wave and it may confuse the interpretation of the electrocardiograms. At first glance the tracings in Figure 70 as represented by







indicative of a smouldering rheumatic fever. Another patient (Fig. 68) merely had a slight "cold" and a questionable murmur in the heart. The markedly delayed conduction ( $P-R = 0.32$  second) meant that the child was suffering from a mild rheumatic fever. More important still was the case illustrated by Figure 69. Here a girl of thirteen was spared an unnecessary operation for appendicitis by the interpretation of the abdominal pain, slight fever and leukocytosis as rheumatic. The third major cause of conduction defects is chronic organic heart disease, especially coronary artery sclerosis and less frequently rheumatic valvular disease (Fig. 67). Hyperthyroidism and syphilitic disease involving the junctional tissue are also rare causes of heart block. When delayed conduction results from digitalis, it disappears within two or three weeks if the drug is omitted. When rheumatic fever is the cause, it may persist longer but generally clears up entirely (Figs. 69 and 72). However, when it is part of a chronic cardiac problem it remains indefinitely and may become more marked, eventually leading to complete heart block.

Although first degree heart block is easily recognized in the electrocardiogram it is difficult and often impossible to detect it clinically. The



Fig. 71.—Heart Block, Second Degree or Partial Heart Block. Note that the conduction time ( $P-R$ ) first rapidly increases, then remains prolonged and finally a beat is blocked; the process repeating itself. The patient had advanced heart failure with hypertension and was on full digitalis dosage when these tracings were taken.

heart remains regular and the rate is often essentially normal. Unless the possibility is kept in mind, there may be no suspicion that a defect in conduction is present. There are two auscultatory findings that may lead one to the correct diagnosis, *i.e.*, the presence of a gallop rhythm or a decrease in the intensity of the first heart sound. It may be that auricular systole is so far removed from the oncoming ventricular beat that it becomes audible or auricular contraction produces some other effect on the valves or muscle which results in a sound. This finding should make the examiner particularly suspicious if it occurs during or following an acute infection. Of course, gallop rhythm is much more common in other conditions in which the  $P-R$  interval is normal. Nevertheless, it may direct attention to an important disturbance that otherwise would be overlooked. Similarly if the loudness of the first heart sound has been noted to decrease during the days of observation, it may mean that the  $P-R$  interval has lengthened.

**Second Degree Heart Block (Partial Heart Block).**—When the defect in conduction is greater, impulses from auricles to ventricles find it increasingly difficult to pass through the junctional tissue until finally one is blocked. This condition is called *partial heart block* (Fig. 71). This may



occur once every seven or more beats or much more frequently, *i.e.*, every second beat (Figs. 72, 73, 74). In fact, more than one beat may be blocked before the next one goes through. These various degrees of

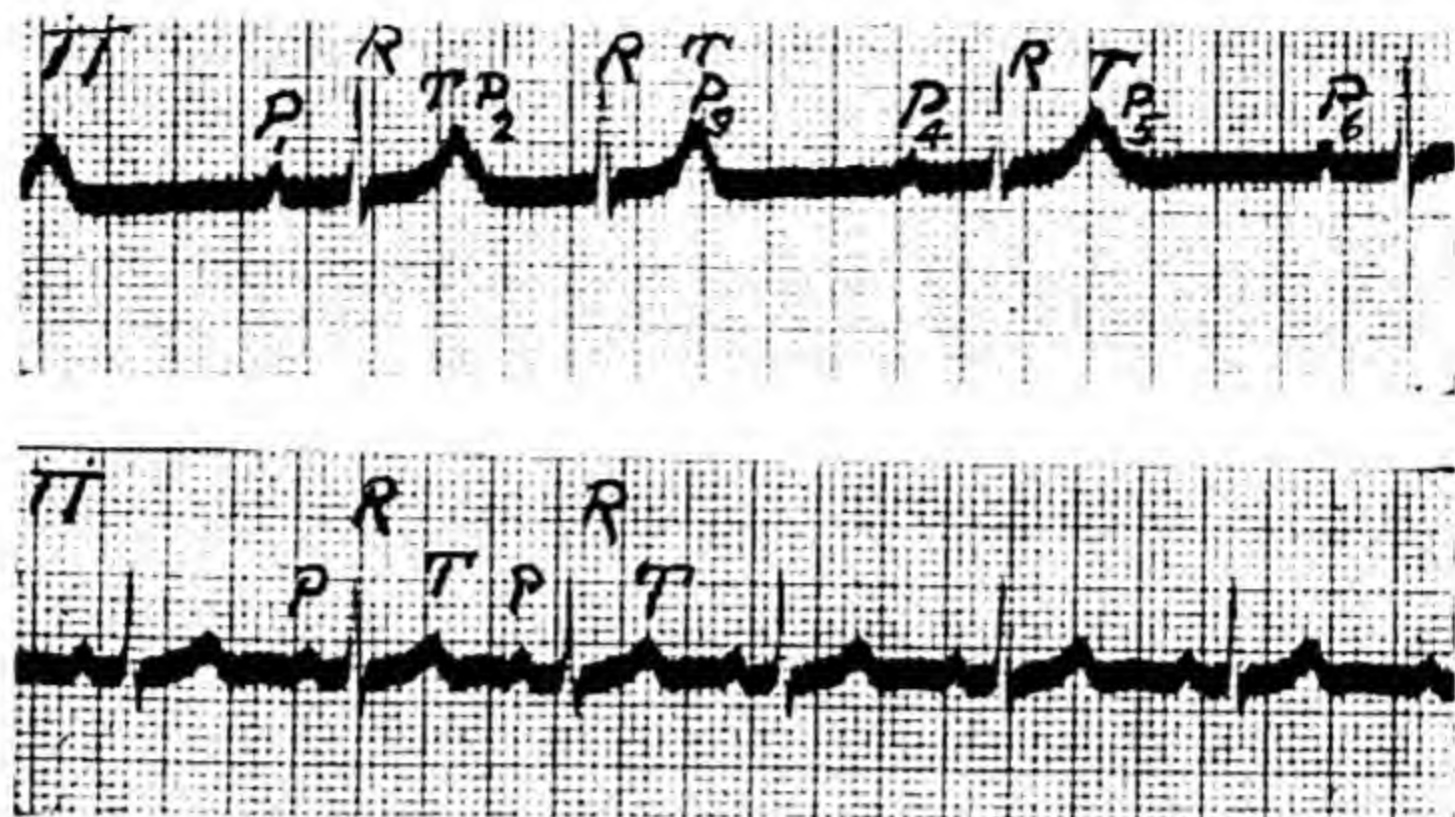


Fig. 72.—Heart Block, Second Degree. The upper tracing was taken June 25, 1929, during acute rheumatic fever; the lower, October 20, 1930, when the patient was well and showed no evidence of heart disease. Note that  $P_3$  and  $P_5$  are not followed by ventricular beats. The lower curves show that the heart block entirely disappeared.

partial block will be called 7:6, 2:1, or 3:1 respectively depending on the relation of the P waves to the ventricular complexes. The auricular and ventricular complexes are normal in form for that heart because the path which the impulses take is normal. The P waves may have to be

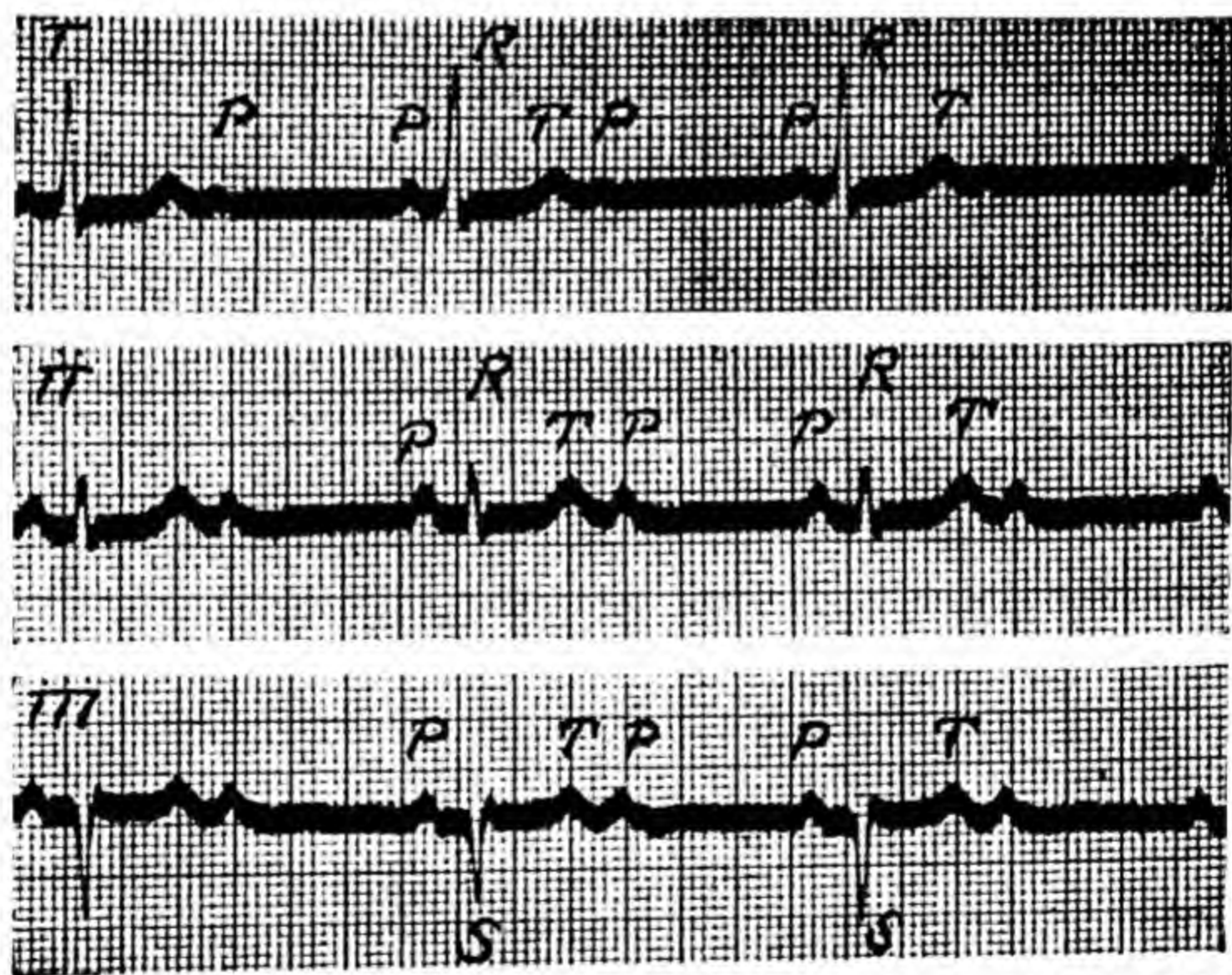


Fig. 73.—Heart Block, Second Degree. Every other auricular beat (P) is not followed by a ventricular beat, *i.e.*, there is a 2:1 heart block. Auricular rate 86; ventricular rate 43. The patient had hypertension, angina pectoris but no syncope.

sought for carefully in a preceding T wave. A slight notching or increase in height of the latter may indicate the presence of the former (Fig. 68, upper curve). The time relation between the two will be abnormal. Figure 71 shows a rapid increase in the P-R interval in the first four



cycles and then the intervals remain the same until a beat is blocked. The increase might have continued but to a slighter degree before the final pause. Because of this diminishing increment in the increase of the P-R interval the heart speeds up before the pause and the shortened P-R interval after the block makes the length of the pause slightly less than two normal heart cycles. (Wenckebach's phenomenon.)

It is not difficult to recognize partial heart block at the bedside. When only an occasional beat is blocked an essentially regular rhythm will be heard and then a sudden pause that will be slightly shorter than two normal cycles. A small auricular wave may be observed in the jugular veins during these pauses. It must be clearly distinguished from extrasystoles for in both conditions the pulse is intermittent. With the latter

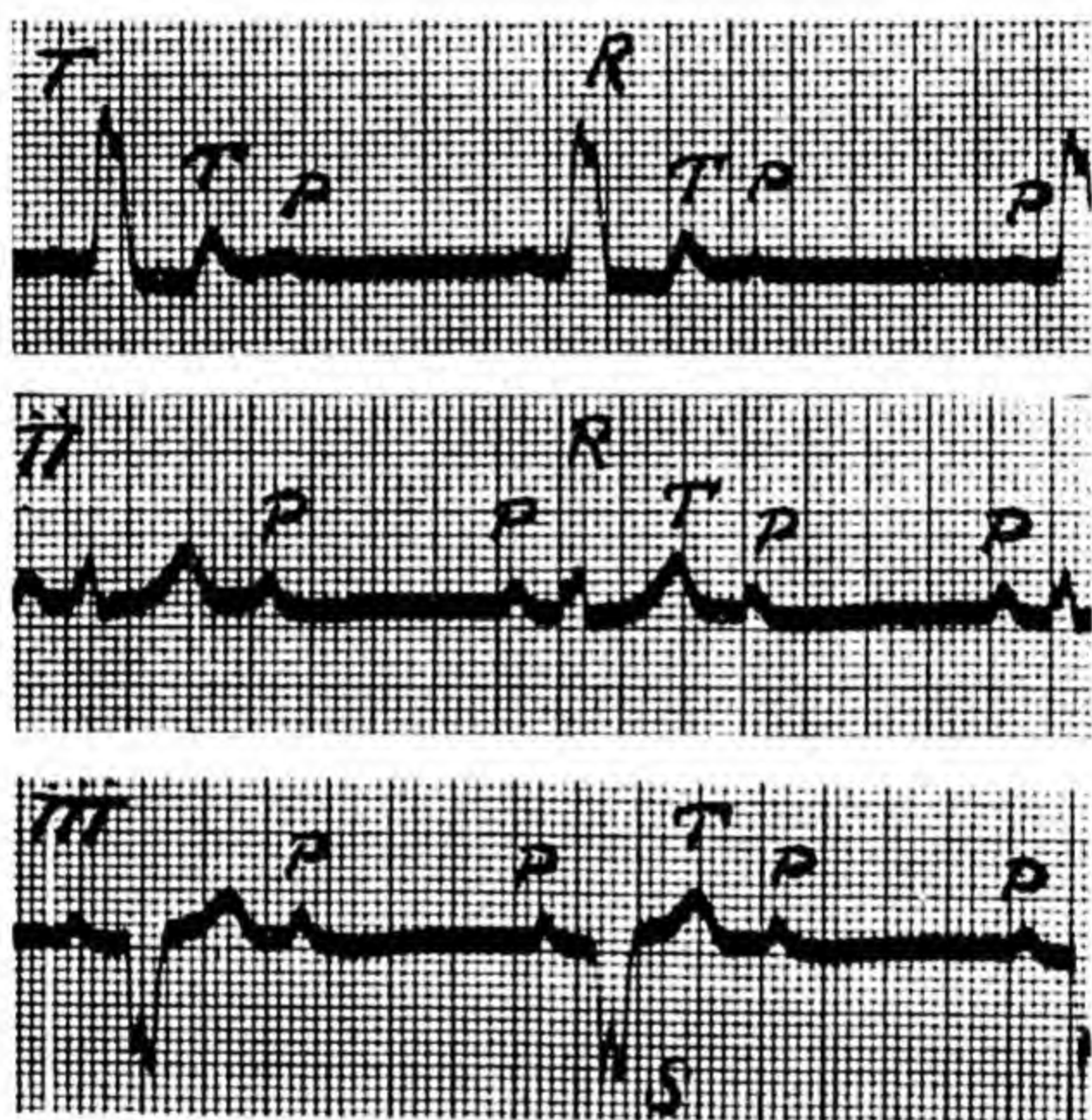


Fig. 74.—Heart Block, Second Degree. Every other auricular beat (P) is blocked (2:1 heart block). Auricular rate 80; ventricular 40. The ventricular complexes (QRS-T) indicate bundle branch block in addition. The patient had hypertension and fainting attacks.

an extra clicking beat will be heard in early diastole. On rare occasions I have confused this sound for the faint sound made by auricular systole in partial heart block. When there is a 3:2 block a coupled rhythm results and this may be misinterpreted as due to some other mechanism. If every second beat is blocked (2:1) a slow regular rate of about 40 is the result. It will need to be distinguished from a normal bradycardia and complete heart block. This often can be done by detecting auricular impulses in the jugular veins and trying to disturb the rate by exercise, breathing, carotid sinus pressure or drugs such as atropine or amyl nitrite. The effect in normal bradycardia will be most marked and gradual; in complete heart block the rate will be unaffected or only slightly changed, the rhythm remaining regular; with partial block abrupt



changes in the length of the heart cycle may result. Atropine or amyl nitrite may lessen the degree of partial block by diminishing vagal tone, and vagal stimulation may increase the block and slow the heart further. Careful attention to the physiologic functions involved and to the exact length of the heart cycles as determined by auscultation will often suffice to differentiate the cause of the slow heart rate.

Partial heart block generally requires no specific treatment. When it accompanies rheumatic infections, treatment is directed at the underlying disease. It merely indicates that a longer period of convalescence is desirable but the prognosis is ordinarily favorable. If it follows digitalis, no harm results. It serves as a warning that the dose of the drug should be lessened. When it is present with chronic organic heart failure, one need not be afraid to give digitalis. Specific treatment will be required if syncopal attacks are threatening. This will be discussed under complete heart block. One might expect that atropine would be of aid in partial heart block by inhibiting the vagus, and occasionally it is very effective in eliminating the block entirely and doing away with the accompanying symptoms (Fig. 75).

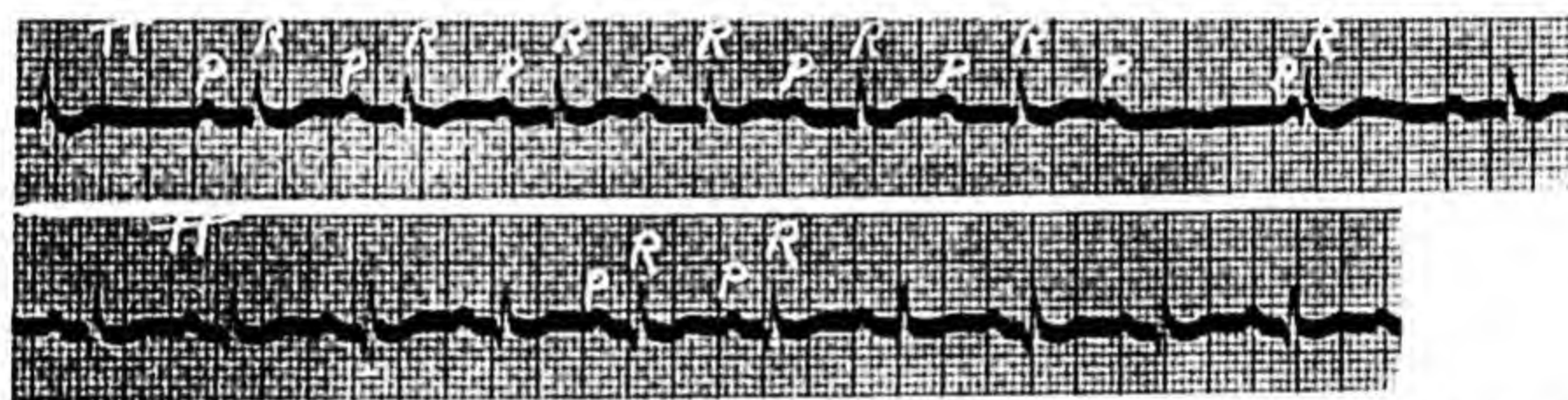


Fig. 75.—Second Degree Heart Block Relieved by Atropine. The upper tracing shows gradual lengthening of P-R interval from 0.21 to 0.32 second and final blocking of a beat. The lower curves taken five minutes after the intravenous injection of 1.0 milligram of atropine show regular rhythm, P-R = 0.17 second.

*Third Degree Heart Block (Complete Heart Block).*—The highest degree of block occurs when the defect is sufficiently great to prevent all impulses from reaching the ventricles, *i.e.*, complete heart block. In this case the auricles contract in sequence to their own pacemaker in the sino-auricular node and the ventricles establish their own rhythm in sequence to a pacemaker just below the defect, either in the lower portion of the a-v node or in the bundle of His (Figs. 76 to 82). Inasmuch as the inherent rhythmicity of the ventricles is slow, the rate is generally about 30 and regular. The auricles and ventricles contract independently of each other. Sometimes the P wave precedes the R, at other times it follows it or comes on the T wave. Occasionally the auricular rate may happen to be just about twice the ventricular and an apparent 2:1 heart block will be seen. But if a sufficiently long tracing is followed or an attempt made to disturb the mechanism, a P wave will generally be found to change its relation to the R wave and actually pass it. The complexes have forms that are essentially normal for that particular heart as the spread of the excitation wave is normal through auricles and ventricles.



The Q-T interval is apt to be lengthened because of the slow rate and the increased duration of systole. Although the ventricular rhythm is usually regular, slight irregularities may be present. Curiously, the ventricular contraction may disturb the auricular rhythm possibly by changes in blood supply or through nervous reflexes.

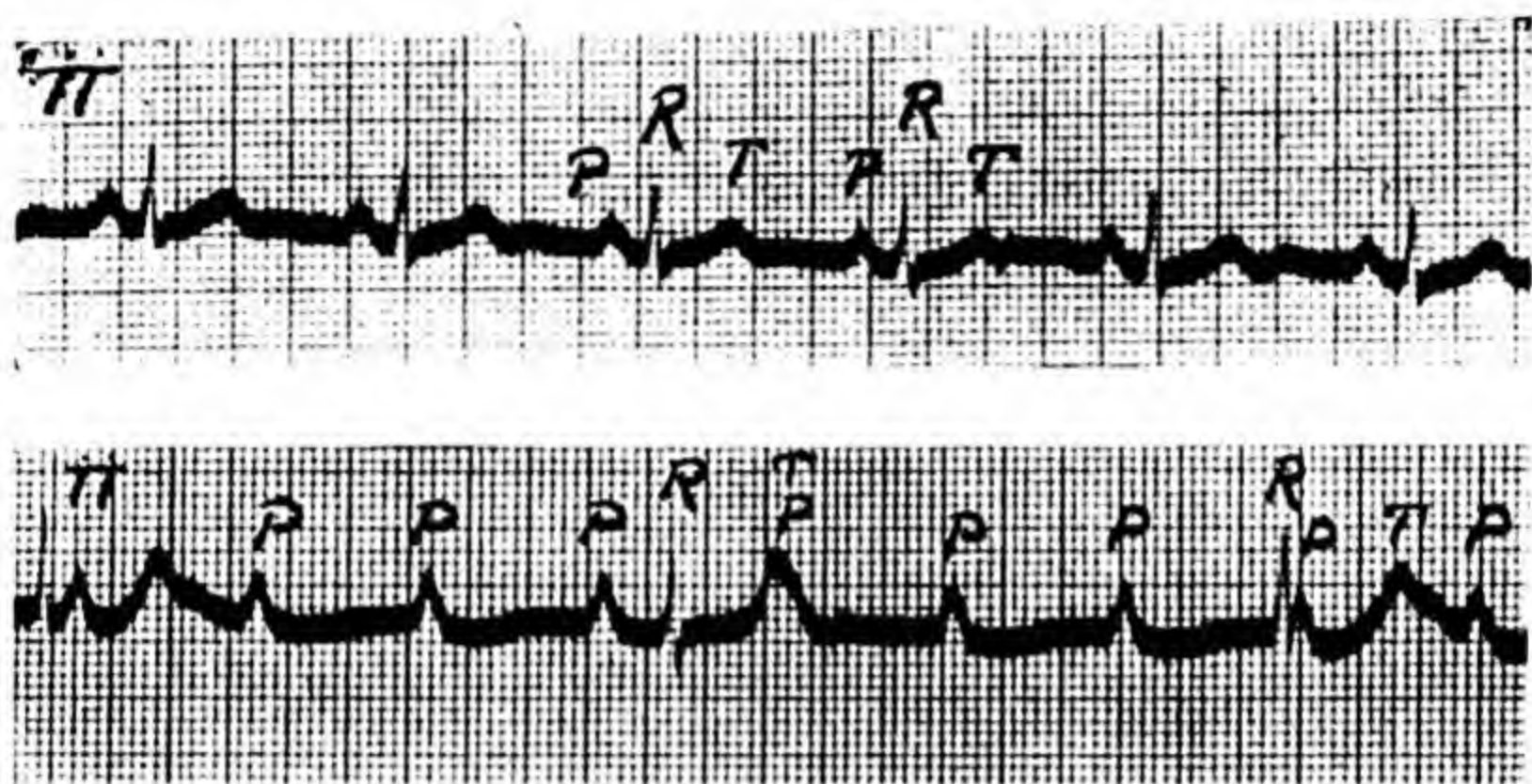


Fig. 76.—Heart Block, Third Degree (Complete Heart Block). The upper tracing, taken September 23, 1931, shows a normal rhythm. The lower tracing, taken May 23, 1933, shows complete dissociation, auricular rate (P) is 103; ventricular (R) is 29. Note the inconstant relationship between P and R waves. The pacemaker of the ventricles is in the a-v junctional tissue. The patient had angina pectoris and Adams-Stokes disease.

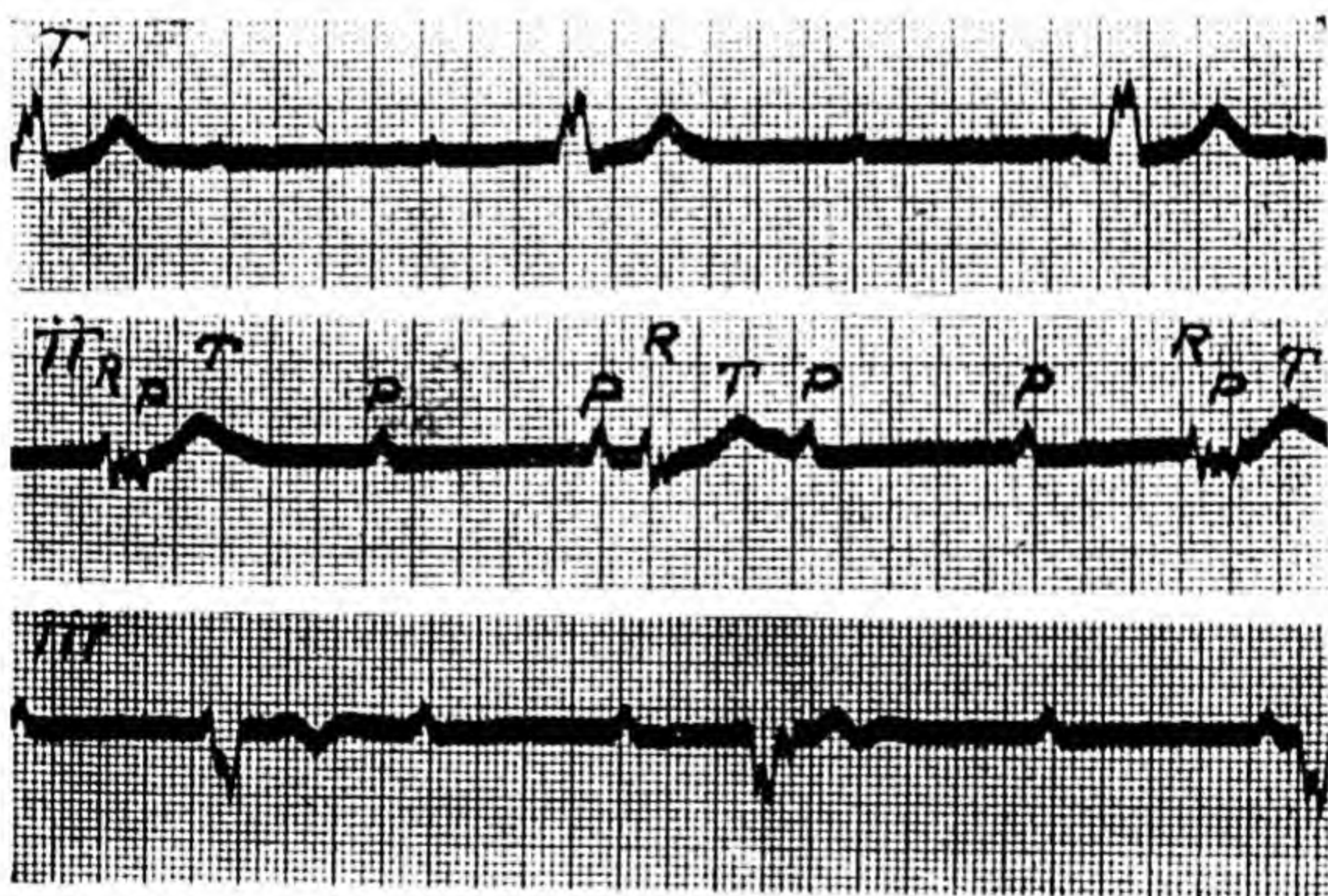


Fig. 77.—Complete Heart Block. Auricular rate 66; ventricular 27. Note that the auricular waves (P) may come just before, during or after the ventricular complexes (R-T). The patient had rare attacks of Adams-Stokes syncope but was able to do hard work.

Although the characteristic ventricular rate in complete block is very slow, rates of 40, 50 and more occur. This is always true when the complete block is due to digitalis (Fig. 42). Higher rates are also found when complete heart block is present in childhood (Fig. 78) and in congenital heart block (Fig. 79). The exact rate probably depends on the



site of the idioventricular pacemaker. When the lesion is higher in the a-v node, the rate will be faster. In general, the lower portions of the heart have more sluggish rates. It is also true that notwithstanding the fact that vagal influences do not affect the ventricles very much, the upper part of the a-v node is somewhat under the influence of the vagus,



Fig. 78.—Complete Heart Block. Auricular rate 90; ventricular 51. Note slight irregularity in the ventricular rate. The patient was a boy, seventeen years old, with probable congenital ventricular septal defect and symptoms of cardiac weakness.

but this effect diminishes at the lower levels of the junctional tissue as the inherent rate decreases.

The most important practical aspect of complete heart block is that with it there is a tendency to syncopal attacks. These attacks are due to further slowing of the ventricles or to complete ventricular standstill. When the rate remains constant, even as low as 30, the efficiency of the

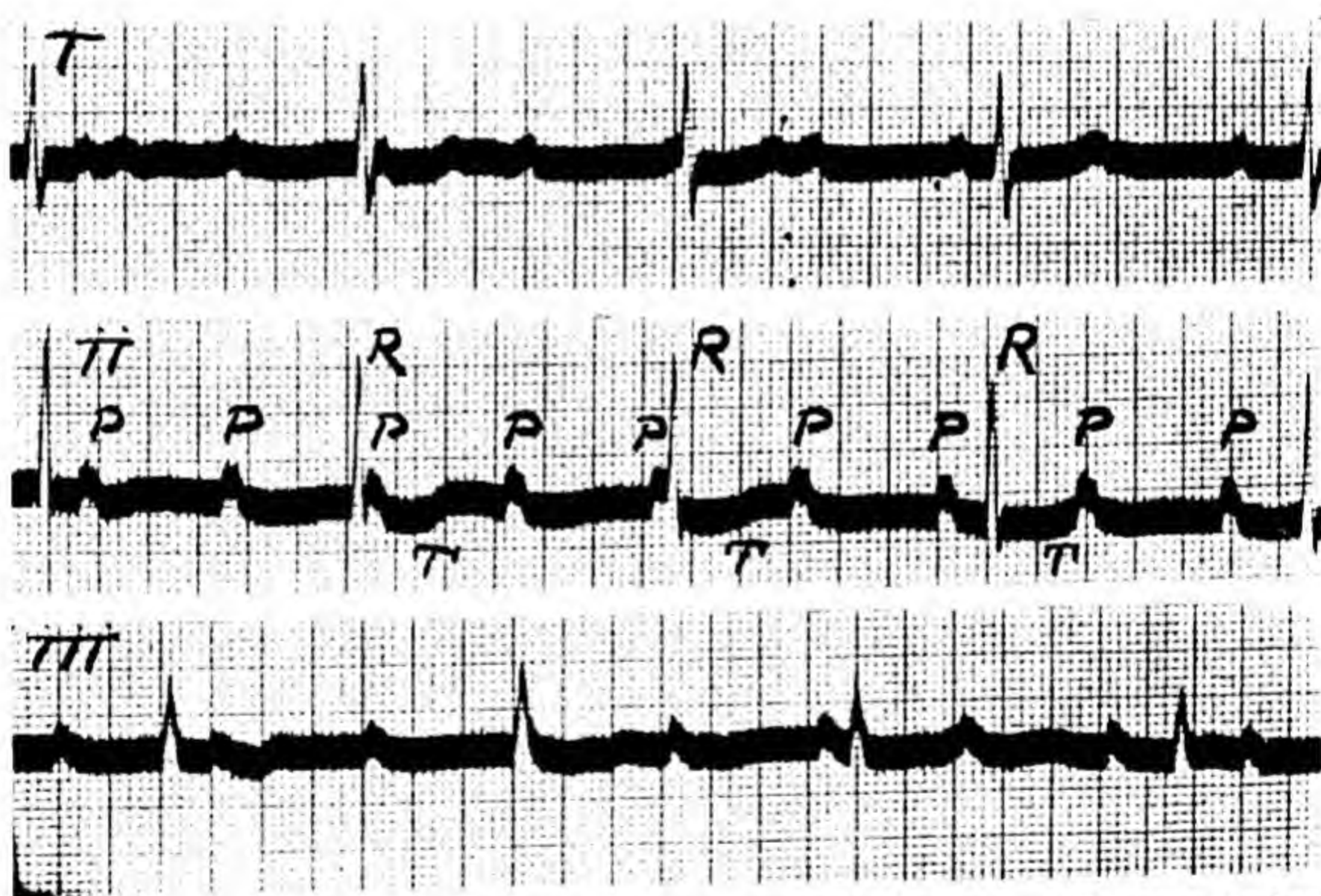


Fig. 79.—Complete Heart Block. This patient was fifty-five years old, known to have had heart block at least since the age of six, probably congenital, and never had any symptoms referable to the heart. Note that the ventricular rate of 44 with congenital heart block is apt to be more rapid than with the acquired type in adults (compare Figs. 76 and 77).

circulation may be normal. I have seen many instances in which patients were able to do hard physical work for many years with such slow hearts. Danger arises when the ventricles stop contracting entirely. Such pauses come suddenly and the symptoms they produce will depend on their length. If the pause lasts several seconds the patients may only feel a faint wave or light-headed sensation like a *petit mal*. If it lasts a little longer, they faint away for several seconds. If it continues for twenty to



sixty seconds they will lose consciousness and have a convulsion with stertorous breathing. If it lasts a few minutes, breathing ceases and death generally ensues. On very rare occasions the normal beat may be resumed even after the heart has ceased beating for several minutes (Fig. 62). Attacks of syncope may occur at the sudden onset of the complete heart block, before the new ventricular rhythm takes on its function. They may also come at the time of the transition between a partial and complete block or when further depression of the pacemaker occurs and the rate falls below 20 (Fig. 80). When, for some reason, possibly through changes in blood supply or nervous influences, the ventricles, which were contracting regularly at the rate of 30 or 20, suddenly stop entirely syncope will occur. Finally, if a patient with complete block develops an attack of paroxysmal ventricular tachycardia, it is apt to be followed by complete standstill of the ventricle (Fig. 62). The precarious feature of complete heart block is, therefore, not the slow regular rate of 28 but the temporary complete failure of the idioventricular pacemaker to send out impulses. Similar syncopal attacks may occur even without

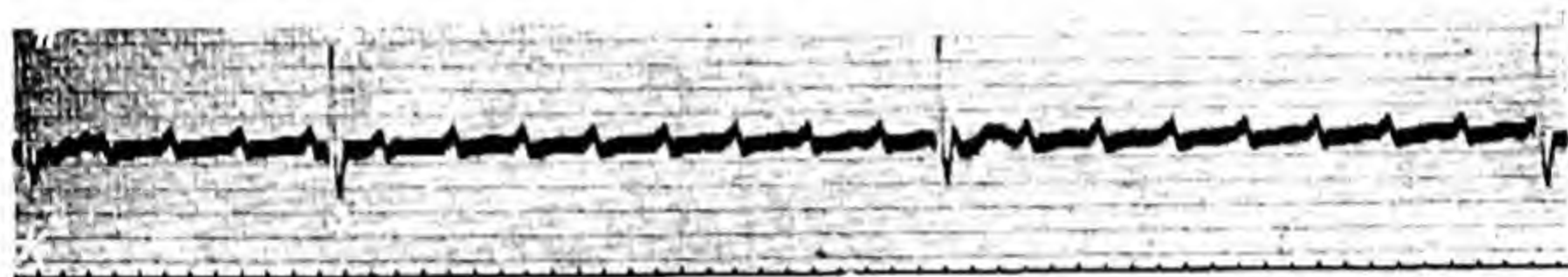


Fig. 80.—Complete Heart Block. The auricles (P) are beating regularly at a rate of 111; the ventricles are contracting slowly and irregularly at a rate of about 16. The two rhythms are independent of each other. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

complete a-v block if the ventricles fail to contract, as may take place reflexly from a sensitive carotid sinus or in sino-auricular block.

The clinical recognition of complete block is ordinarily a simple matter when the rate is very slow and regular as there is no other condition in which this occurs. When the rate is between 35 and 50 or more it can be confused with other conditions, especially partial block. The differentiation has already been discussed in the paragraphs on partial heart block. There is one additional sign that is pathognomonic of complete block, *i.e.*, the changing quality and intensity of the first heart sound. On careful auscultation it will be noted that with occasional cycles the first heart sound may become muffled, accentuated or reduplicated. This results from the changing relationship between the auricular and the ventricular contractions. In some cases the auricular rate is exactly twice the ventricular for long periods of time so that only after prolonged auscultation will this valuable diagnostic sign become apparent (Fig. 81). Furthermore, it is often possible to hear extra-auricular sounds over the precordium during the long pauses, to see extra-auricular waves in the jugular pulse and even to feel faint waves in the radial pulse synchronously with auricular systole which are due to an impact against the aorta. All these latter signs merely indicate that the auricles are contracting



more frequently than the ventricles and that there is some degree of block. The changing quality of the first heart sound and other criteria prove that the block is complete.

The clinical conditions in which complete heart block occurs were taken up under partial heart block. Suffice it to mention that it is found associated with toxic doses of digitalis, certain infections, notably rheumatic fever and diphtheria, and certain cases of chronic heart disease. In the latter condition, once established, complete heart block is apt to be permanent, although occasional reversion to a block of lesser degree or a normal mechanism occurs.

Although the majority of cases of heart block associated with attacks of Adams-Stokes syncope will show a slow heart and complete block between spells there is a considerable number of patients who have a normal rate and no block except when they are having attacks. It fol-

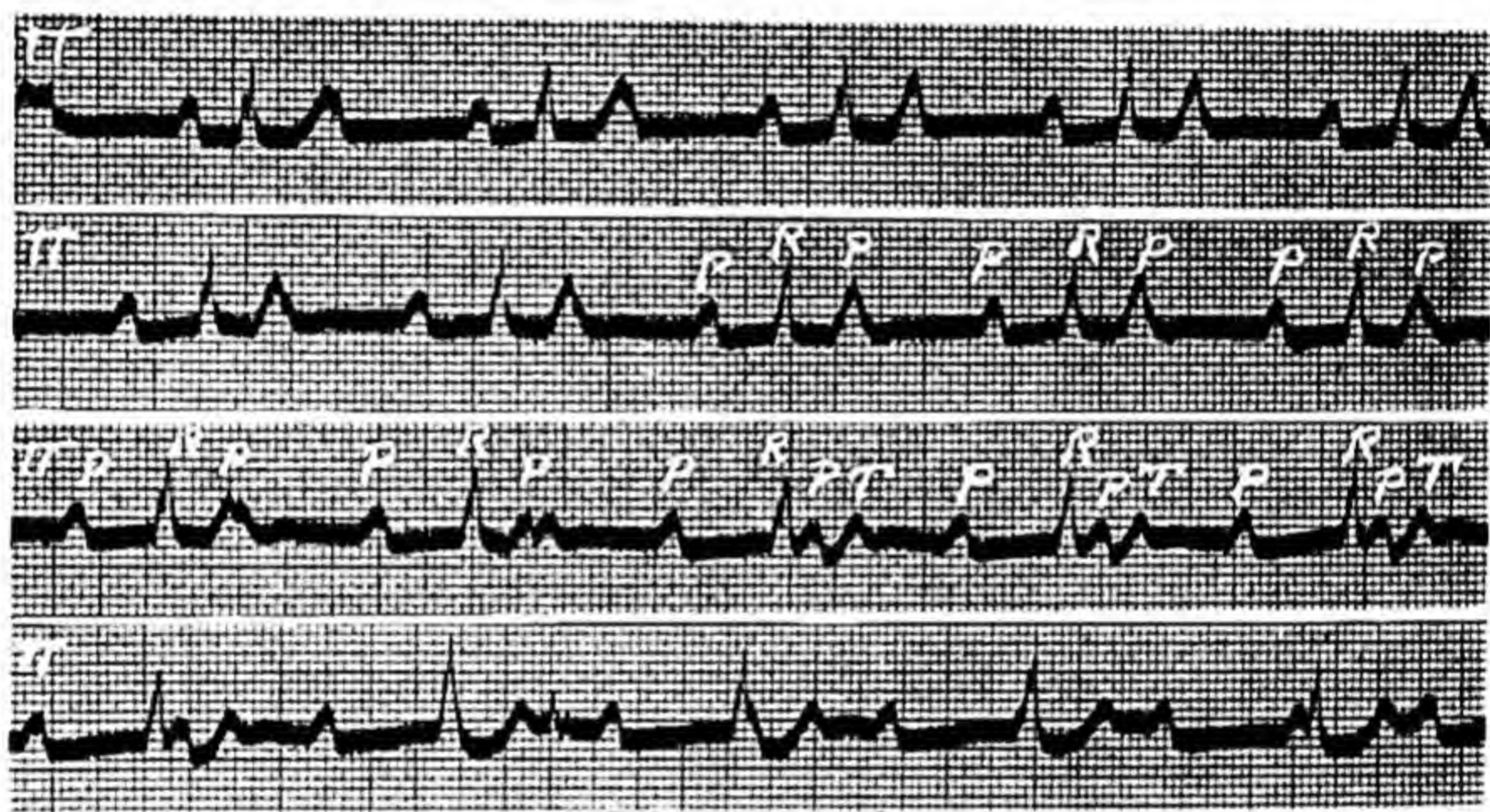


Fig. 81.—Complete Heart Block. Continuous tracing shows that while for a long period (upper two strips) a 2:1 relationship appears to exist, complete dissociation finally develops (lower two strips) with the P waves crossing the R waves.

lows, therefore, that finding a normal rhythm in a patient with fainting spells by no means rules out the diagnosis of Adams-Stokes attacks (Fig. 82).

Therapy in complete heart block is important because it is often effective. Treatment is not directed at the slow steady rate, for this may be productive of no symptoms and may even be a protection to a heart that is threatened with congestive failure. However, the slow rate is no contraindication to the use of digitalis if congestive signs are present. The main concern is the attacks of syncope. When they occur at rare intervals, once every year or more, the problem is difficult because preventive medication would have to be continued all this time and it might even then be doubtful whether or not the cessation of attacks was due to the drugs. When attacks occur frequently, many a day, as may happen during acute coronary thrombosis, adrenalin given subcutaneously may be life saving. It may be necessary to give a dose of 0.3 to 0.5 c.c. of a



1:1000 solution every two hours for a day or so. The frequency and duration of the treatment will depend on the circumstances. For a period of an hour or more after adrenalin is injected all attacks may cease. The drug increases the irritability of the ventricles and not only prevents the pauses of the ventricles but actually increases their rate in complete heart block. When the condition is chronic and the attacks occur in an otherwise ambulatory patient, ephedrine sulfate administered by mouth (0.025 gram— $\frac{3}{8}$  grain—or even larger doses) two or three times a day may prove effective. Occasionally barium chloride (0.03 to 0.06 gram— $\frac{1}{2}$  to 1 grain—three or four times a day) given by mouth will inhibit all

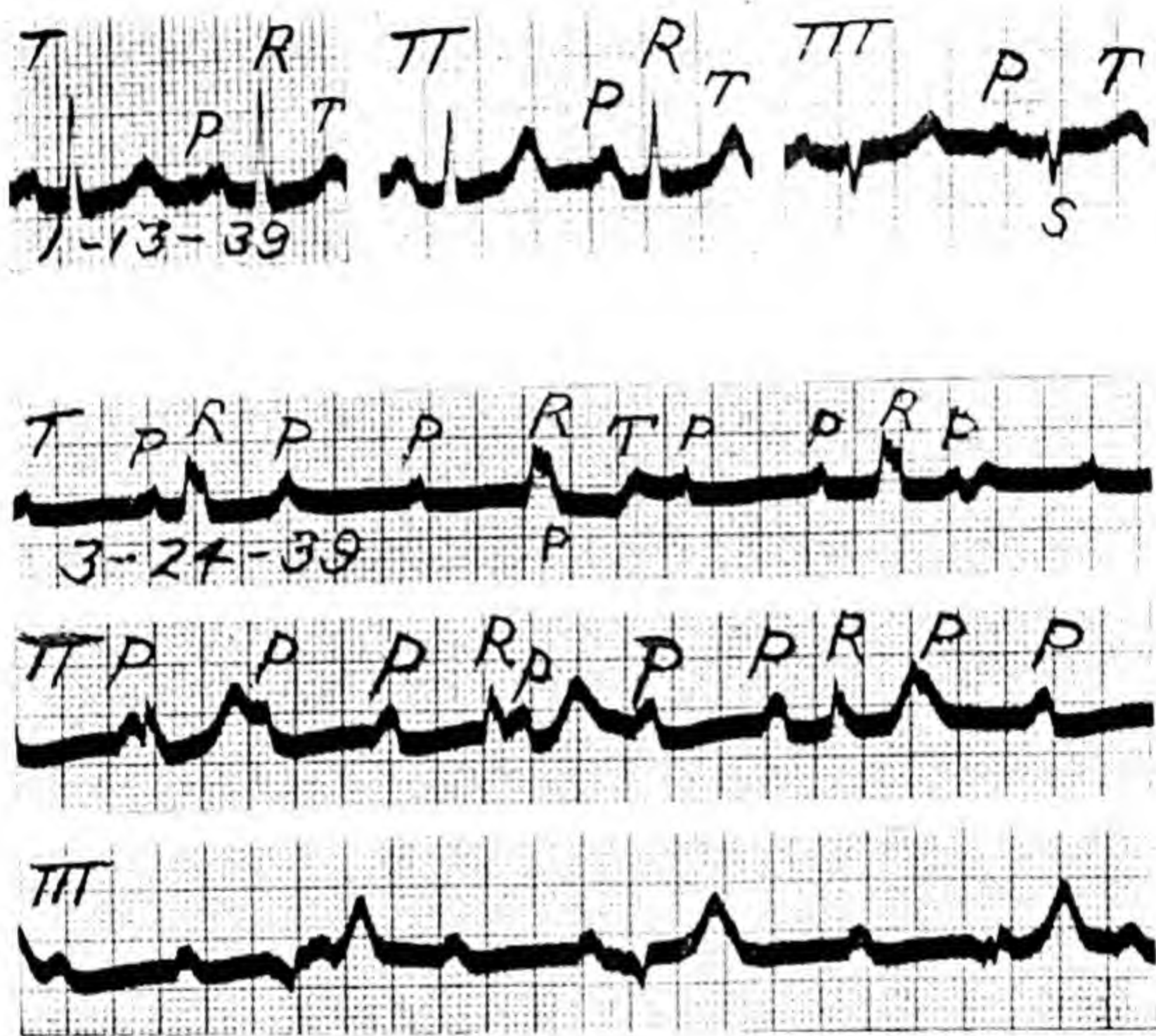


Fig. 82.—Complete Heart Block. (Adams-Stokes attacks with normal rhythm between spells.) The patient was a woman sixty years old with hypertension and mitral stenosis. At the time the first tracing was taken she already had had frequent attacks of unconsciousness. Two months later she showed typical complete block and syncopal attacks continued.

attacks. In stubborn cases a great variety of procedures have been employed and when an apparently favorable result has been obtained it is difficult to be certain of the efficacy of the agents employed in treatment. Amongst these are inhalations of 1:100 adrenalin, benzedrine, propadrine, full doses of atropine, intravenous injections of 50 per cent glucose, thyroid extract, cardiazol, and digitalis or uarginin. It must be remembered that quinidine should not be given to patients with disturbances in conduction as it can increase the inhibition of beats.

**Bundle Branch Block.**—An impulse may start in the normal sinoauricular node, travel across the auricles, continue down the auriculoventricular node and bundle of His and yet be blocked in the right or left



main branch of the bundle. If the block occurs in the left branch, the impulse descends the right branch normally and reaches the left ventricle in a roundabout fashion, probably through the ventricular septum. The reverse process occurs if the right branch is blocked. The ventricles continue to contract regularly so that there is no arrhythmia in the ordinary sense. One ventricle, however, responds slightly ahead of the other. The ventricular complexes will necessarily be abnormal because of the circuitous route taken by the impulse. In fact, they will resemble a series of ventricular extrasystoles arising from the unblocked side or the curves seen in ventricular tachycardia. The QRS complex is broadened, coarsely notched, frequently of considerable amplitude and the T wave is apt to

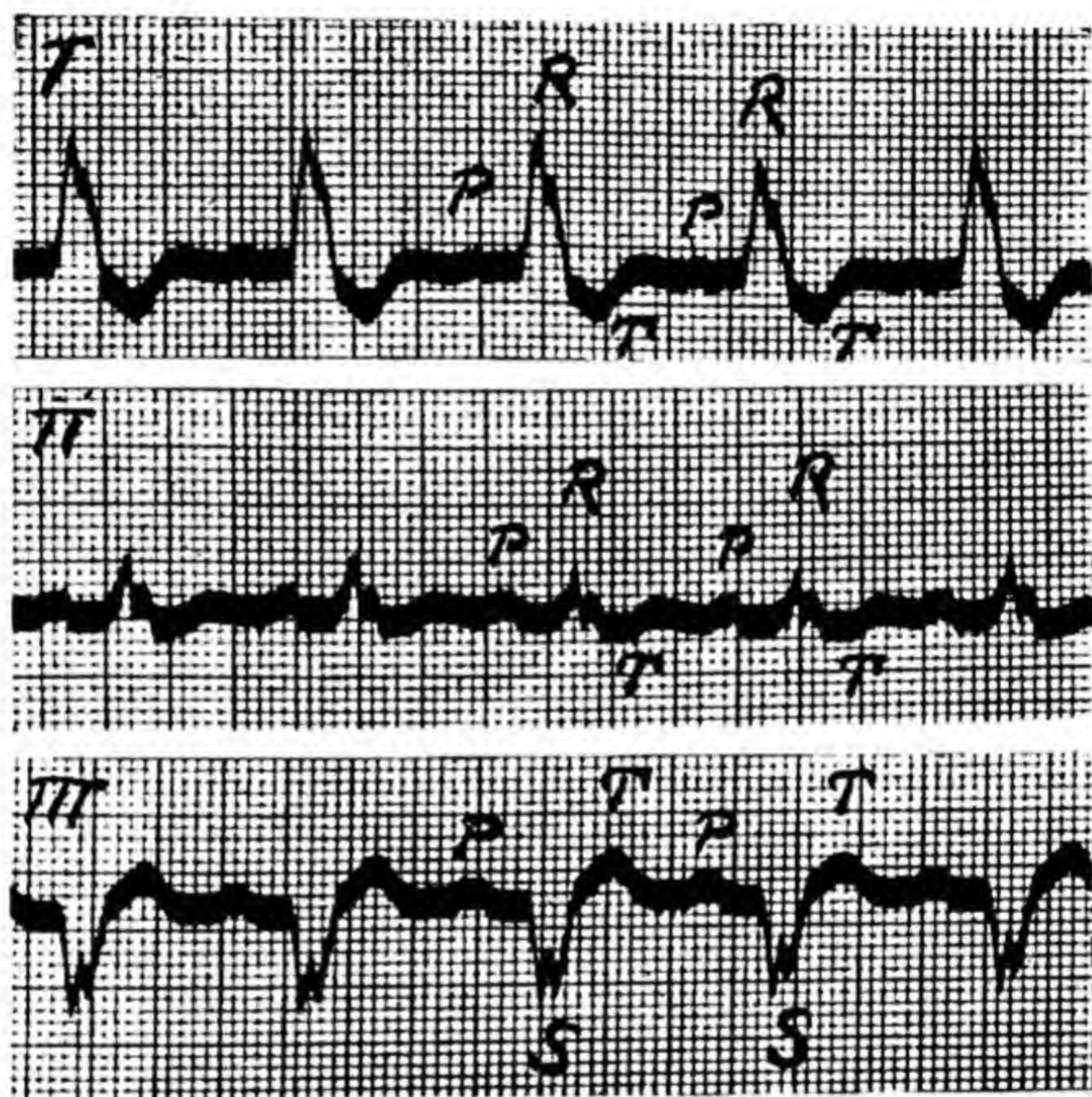


Fig. 83.—Left Bundle Branch Block (new terminology). Note that the initial ventricular complexes (QRS) are broad, measuring 0.15 second (normally about 0.08 second), the QRS waves are coarsely notched and the T waves are in the opposite direction to the main initial deflections in Leads I and III. Such curves indicate block of impulses in the left branch of the bundle of His. The rhythm of the heart is regular. This patient had calcified aortic stenosis with marked cardiac failure.

continue quickly in the opposite direction to the main initial deflection. When the initial deflection is upward in Lead I and downward in Lead III the curves represent *left bundle branch block* (Figs. 83 to 86) and when it is downward in Lead I and upward in Lead III it is *right bundle branch block* (Figs. 87 to 89). Lead I is more important than the other two leads in determining whether the block is in the right or left branch. If there is a prominent S wave in Lead I, the block is on the right side; if there is a prominent R and no S, it is on the left. This is the new terminology which is now generally accepted and is the reverse of that first proposed.

There must be numerous instances in which there is delay but no block in one branch or the other and this must result in electrocardiograms



that resemble those just described. They may be included in the general group of defective intraventricular conduction which condition will be discussed. When there is doubt concerning bundle branch block, pre-

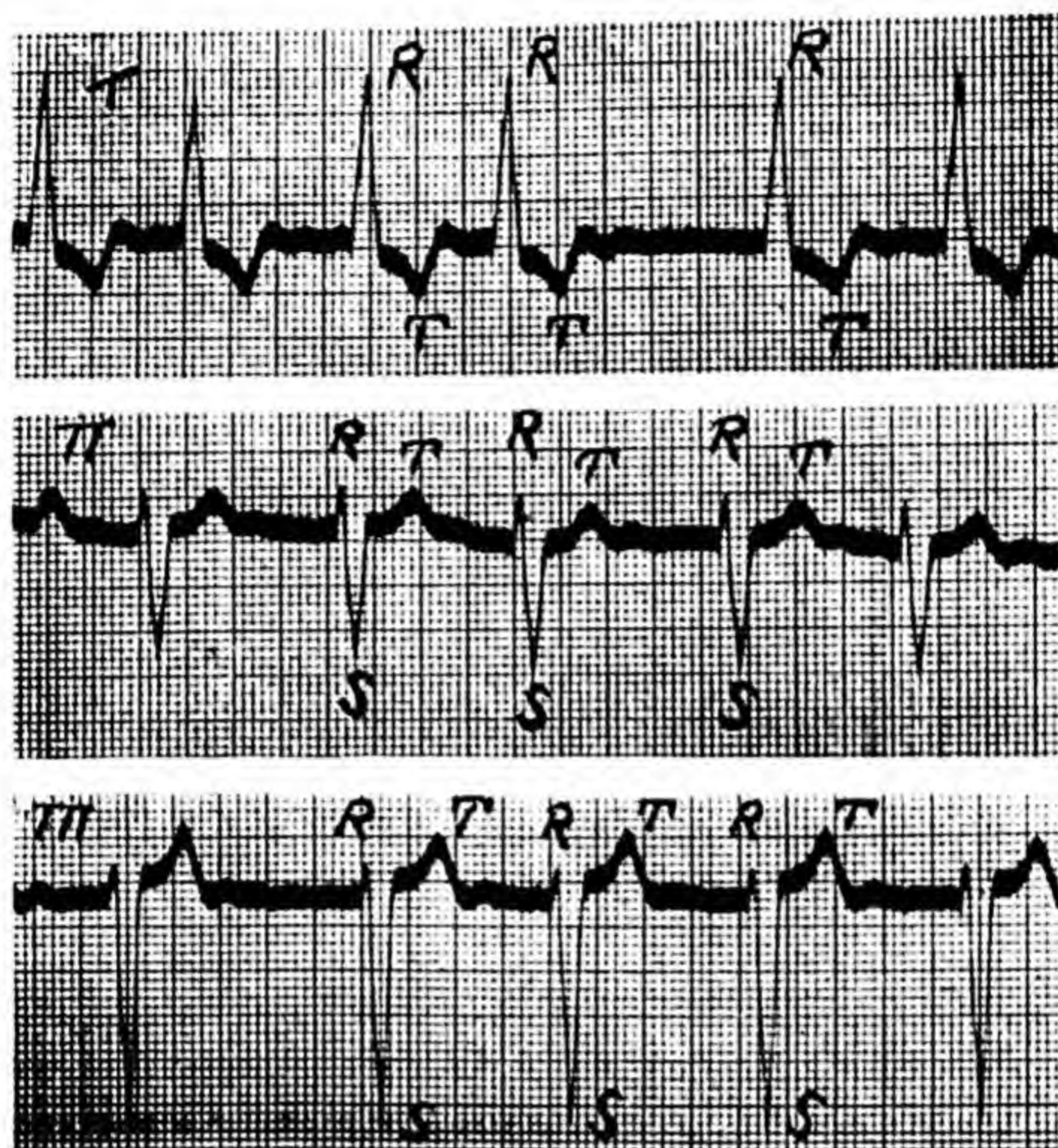


Fig. 84.—Left Bundle Branch Block. Note the prominent, broad and coarsely notched QRS complexes with oppositely directed T waves. In this case the rhythm is irregular because of a coexisting auricular fibrillation. The patient had hypertensive heart disease and chronic nephritis.

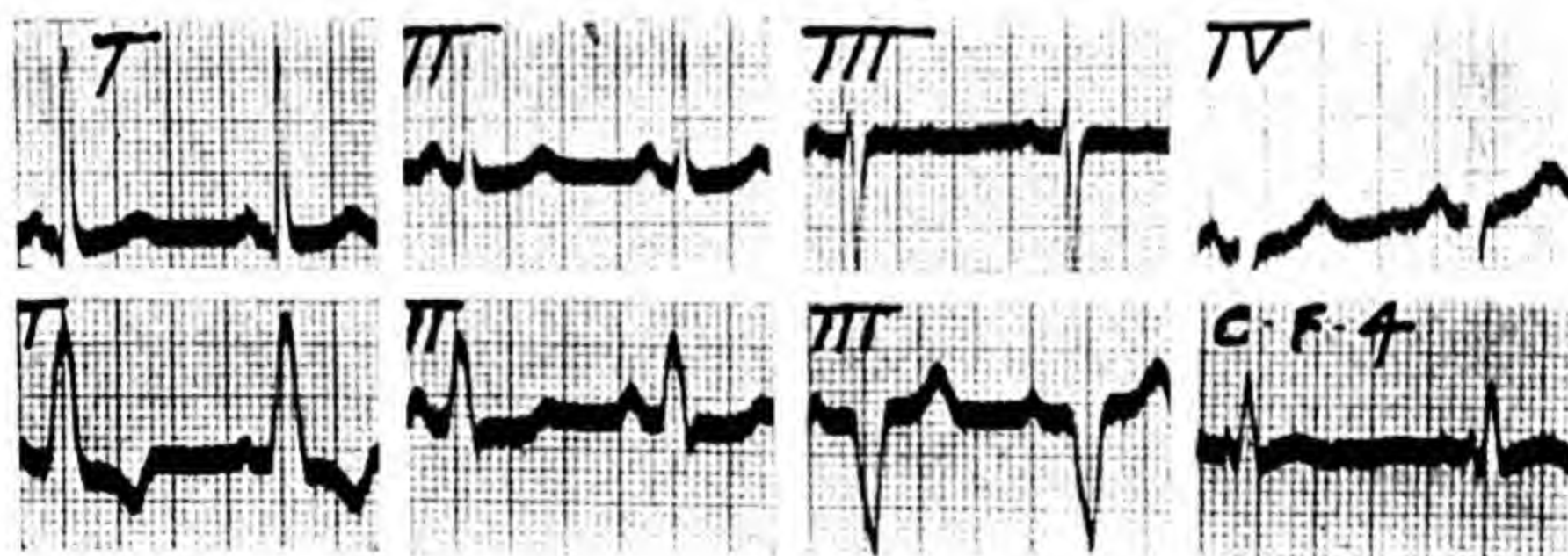


Fig. 85.—Left Bundle Branch Block. The upper tracing (made February 14, 1938) shows left axis deviation with normal QRS interval (0.08 second). The lower curves (February 2, 1940) show QRS = 0.12 second. Note the late downstroke in Lead CF<sub>4</sub>. The patient was a woman sixty-five years old, mainly neurotic, followed for many years, who gradually developed hypertension (blood pressure 200 systolic, 110 diastolic), never had any specific cardiac symptoms and showed little abnormality on examination.

cordial leads (as will be discussed later) will help in the determination of the diagnosis (Fig. 85). Bundle branch block is often associated with other types of heart block such as a-v block or s-a block (Fig. 65). It also



may occur when auricular fibrillation is present (Fig. 84). In Figure 86 is represented a rare instance of 2:1 left bundle branch block. At first glance one might interpret this as coupled rhythm due to premature ventricular systoles, but on careful inspection it will be found that auricular and ventricular beats come exactly on time.

Bundle branch block is fairly common in general practice. Block on the left side is much more frequent than on the right. This is in accord with the general experience that disease of the left ventricle is more prevalent than disease of the right ventricle. Inasmuch as it occurs for the most

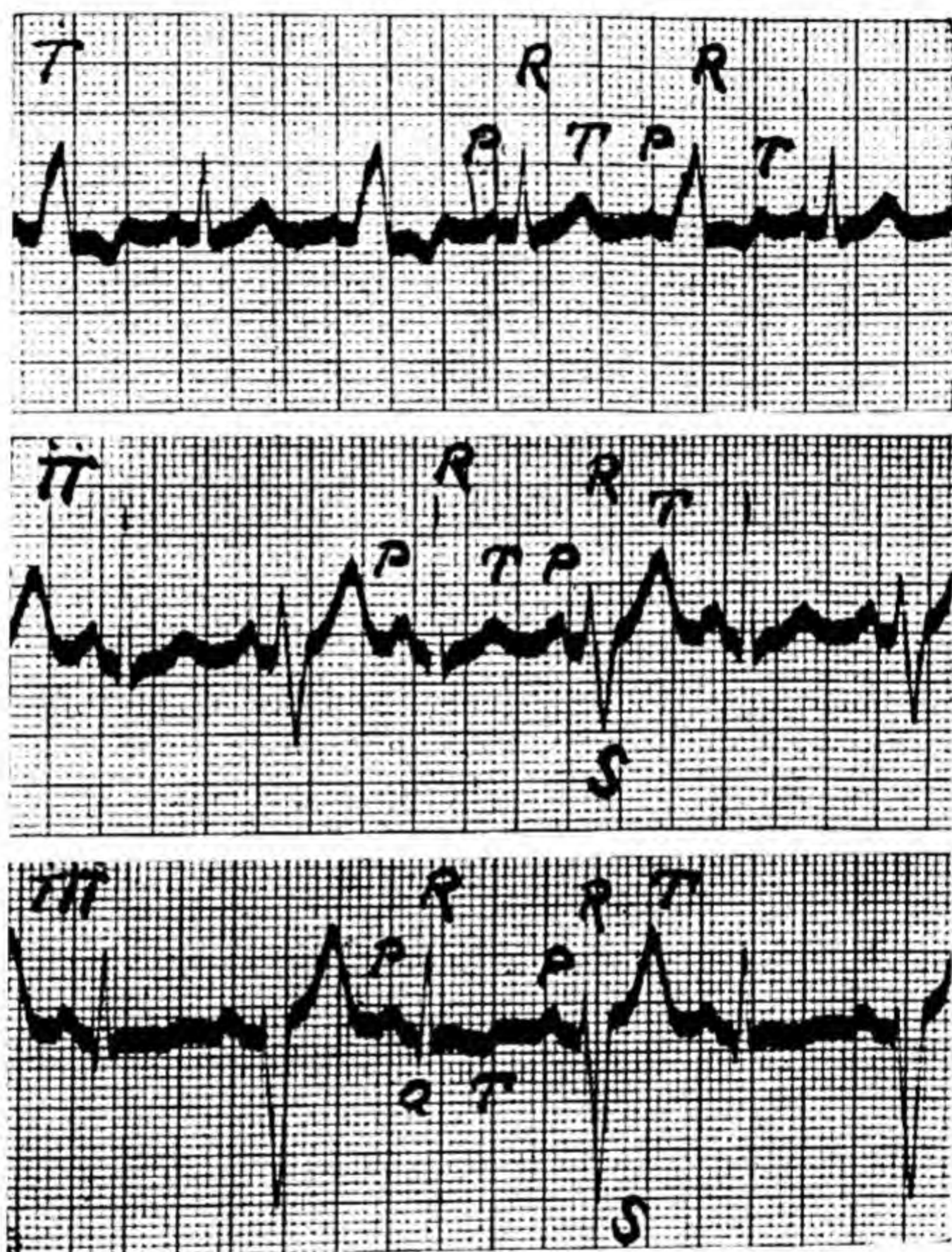


Fig. 86.—Left Bundle Branch Block (2:1). Note that every other ventricular complex has the form similar to Figs. 83 and 84. The other beats are normal. The rhythm is perfectly regular. This indicates that every second beat fails to be conducted down the left branch of the bundle of His. This patient had hypertensive heart disease.

part in patients with a regular rhythm it is difficult to recognize this condition without electrocardiograms. However, it is often possible to suspect its presence. Whenever a diastolic gallop rhythm is heard one should think of the possibility of bundle branch block. Moreover, bundle branch block is often accompanied by a bifurcated or reduplicated apex impulse. Both of these findings need to be looked for carefully and deliberately for when they are detected, bundle branch block is apt to be present. Finally, in such cases pulsus alternans is also very common. In fact, the frequent association of bundle branch block, gallop rhythm and pulsus



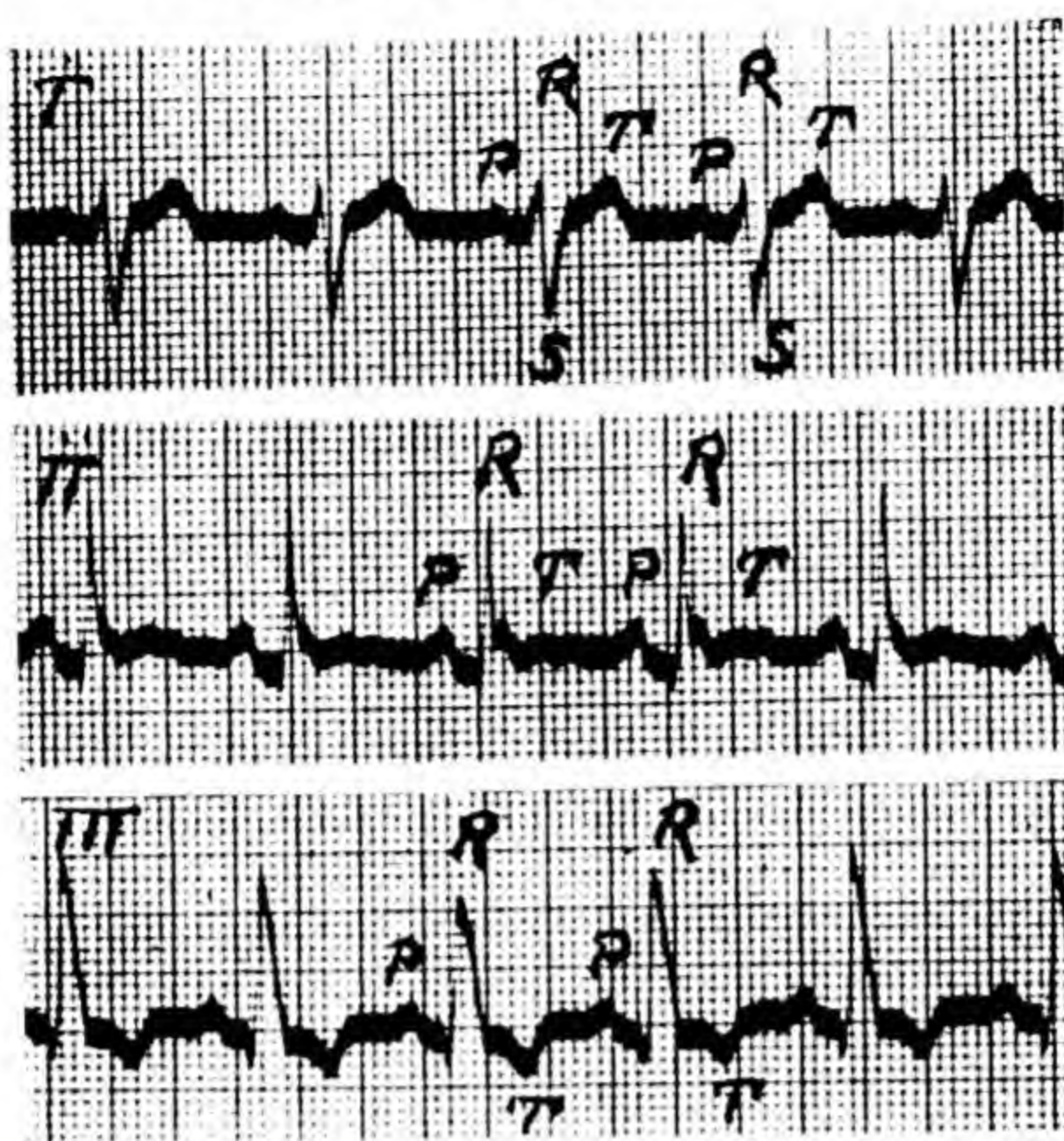


Fig. 87.—Right Bundle Branch Block (new terminology). Note that the general characteristics of the ventricular complexes (QRS-T) are the same as those in Figs. 83 to 86 but that the main deflections are opposite, going downward in Lead I and upward in Lead III. These curves indicate a block in the right branch of the bundle of His. This patient, twenty-two years of age, had one fainting attack but otherwise has no cardiac disability.

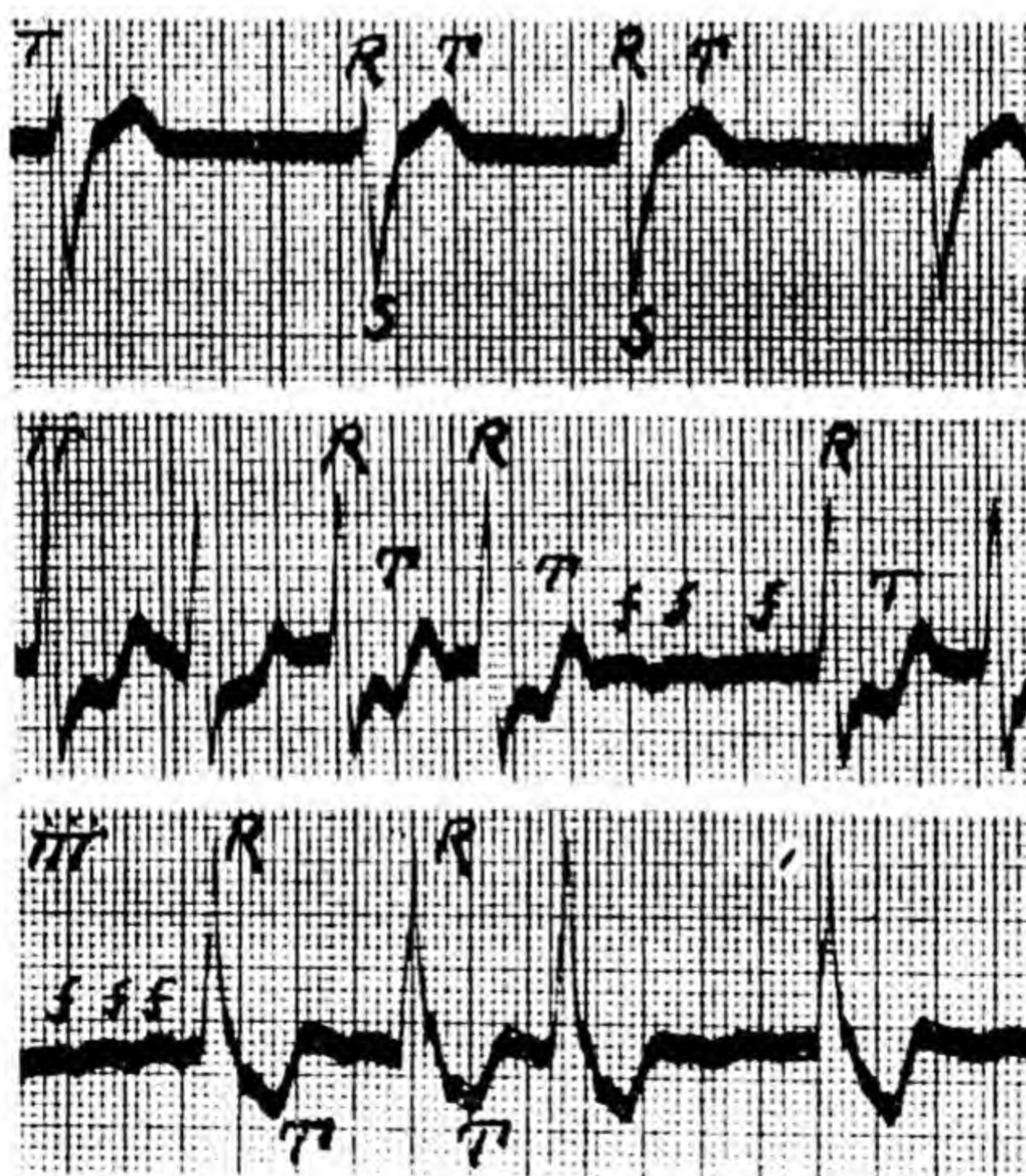


Fig. 88.—Right Bundle Branch Block. The ventricular complexes are similar to those in Fig. 87 but the rhythm is irregular due to a coexisting auricular fibrillation (f-f-f). The patient is seventy-two years old, has had such curves for at least eight years and shows evidence of well-compensated aortic and mitral stenosis and hypertension. He used to have frequent attacks of Adams-Stokes syncope.



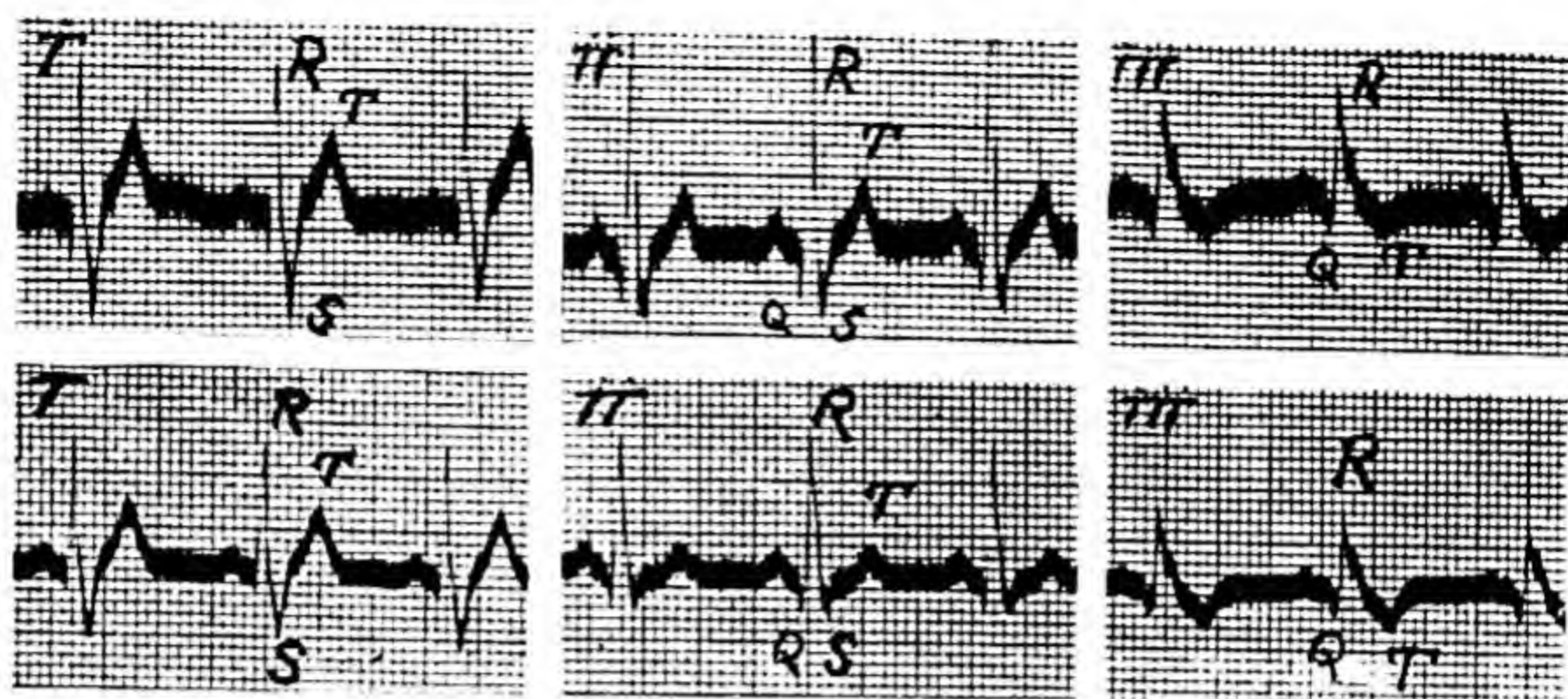


Fig. 89.—Right Bundle Branch Block. The upper tracing was taken August 15, 1919; the lower, July 11, 1933. Note the prolongation of the QRS complex measuring 0.12 second (normally about 0.08 second). These curves are somewhat similar to those in Figs. 87 and 88. The patient at first only had slight hypertension, carried on well for fourteen years and then developed angina pectoris.

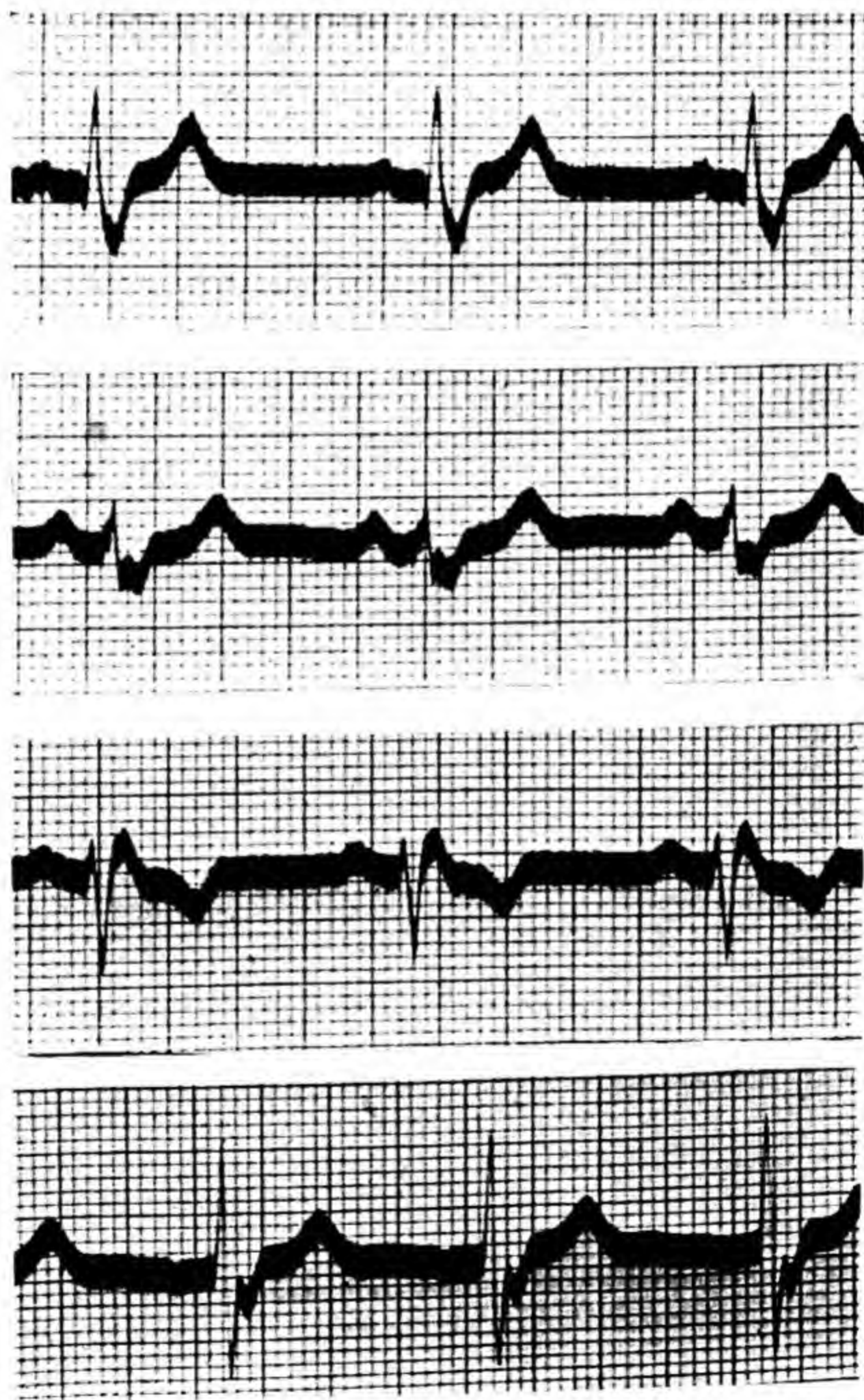


Fig. 90.—Right Bundle Branch Block. Note the spread of QRS complex which measures 0.14 second, a broad  $S_1$  and an early downstroke in Lead IV taken from the apex region (left ventricle). The patient had angina for eighteen years. All the electrocardiograms obtained during this period were similar to this one. The patient died of cancer at the age of sixty.



alternans should lead one to suspect the presence of one if the other two are found.

Bundle branch block occurs most frequently in conjunction with hypertensive heart disease, with disease of the coronary arteries, in cases of aortic stenosis and much less so in association with mitral stenosis. It always denotes some disease of the myocardium. The prognosis for patients with left bundle branch block is poor. The average length of life after it is first noted is not more than a year or two but there are exceptional patients who carry on fairly satisfactorily for many years. The situation is quite different in right bundle branch block, for here many patients continue in good health for a great many years (Figs. 87 to 90). In fact, there are instances of this type in which there seems to be very little other evidence of organic disease and the prognosis may be ex-

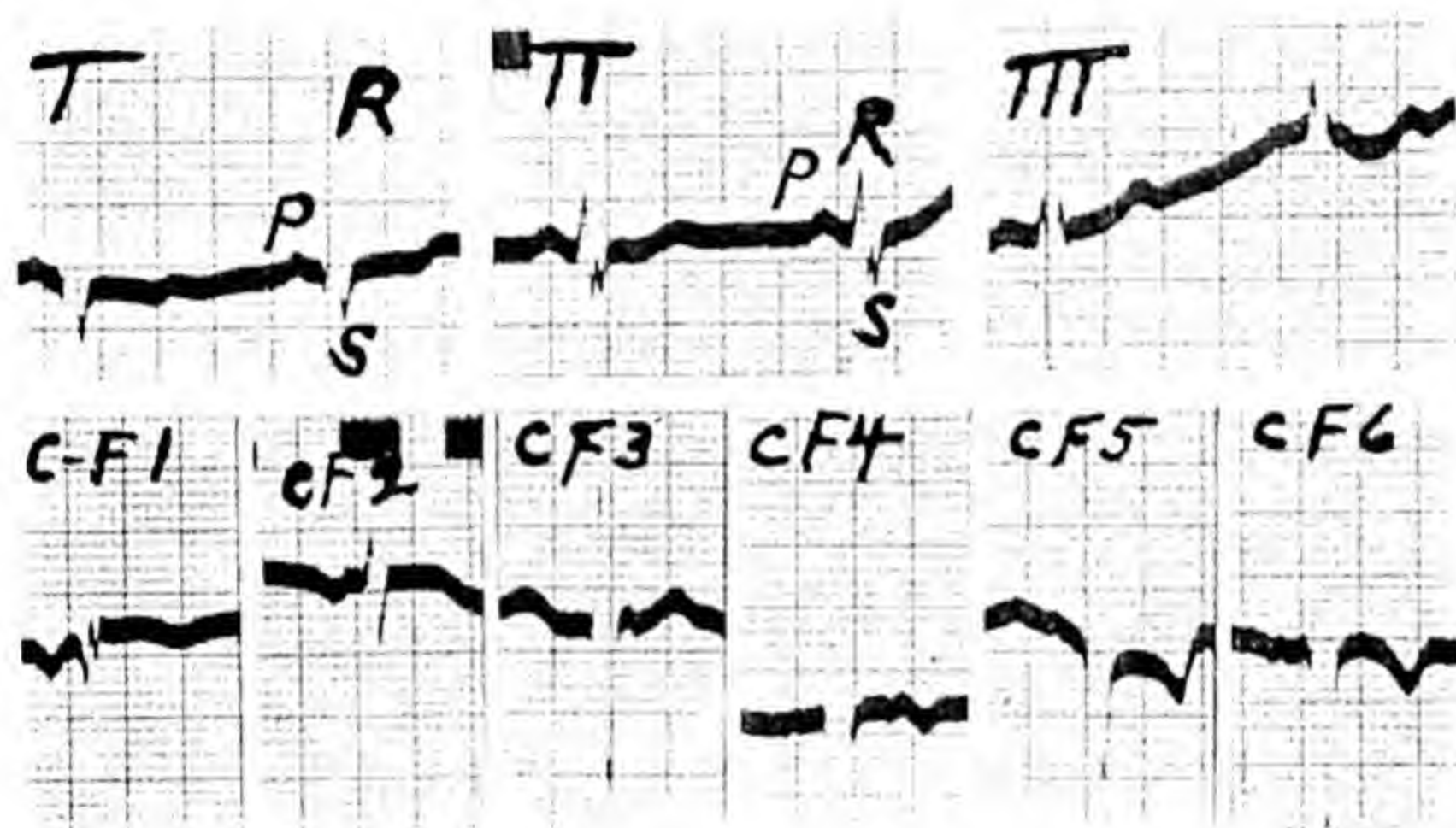


Fig. 91.—Intraventricular Block. The upper tracing shows the three conventional leads. Note that the QRS interval is prolonged, measuring 0.11 second. The lower set was taken from six positions over the precordium coupled with left leg ( $CF_1$  to  $CF_6$ ). The QRS waves are indistinct but there is no late downward deflection (intrinsic wave) over the left side of the precordium ( $CF_4$  to  $CF_6$ ). The patient had hypertension (190 systolic and 100 diastolic), angina pectoris, and prostatic hypertrophy.

tremely favorable. There is no specific treatment for persons with this disturbance as it does not, by itself, produce any handicap. It merely reflects the condition in the heart muscle and therapy is directed at the general state of the circulation.

**Defective Intraventricular Conduction (Intraventricular Block).**—Impulses may be delayed in their passage through either branch of the bundle of His without being blocked (*incomplete bundle branch block*) or they may be blocked in the finer branches of the Purkinje system (*arborization block*). Whether the latter condition actually produces disturbances in the electrocardiogram which have been accredited to it is uncertain and the exact localization of delay in one of the branches is difficult. Both of these possibilities have therefore been included in the general term *intraventricular defective conduction*. The ventricular complexes will neces-



sarily resemble those described in the previous paragraphs. The QRS complex is spread, measuring 0.1 second or more, it is coarsely notched, but the T waves need not be in the opposite direction to the main initial deflection in Leads I and III (Figs. 91, 92). The ventricular complexes are not apt to be as large as in typical bundle branch block. In doubtful cases the distinction between bundle branch block and intraventricular block is best studied in the precordial leads.

The clinical implications of defective intraventricular conduction are similar to those of bundle branch block. It should be borne in mind that generally a grave condition of the ventricular musculature is indicated and the prognosis should be guarded accordingly. Occasionally, such

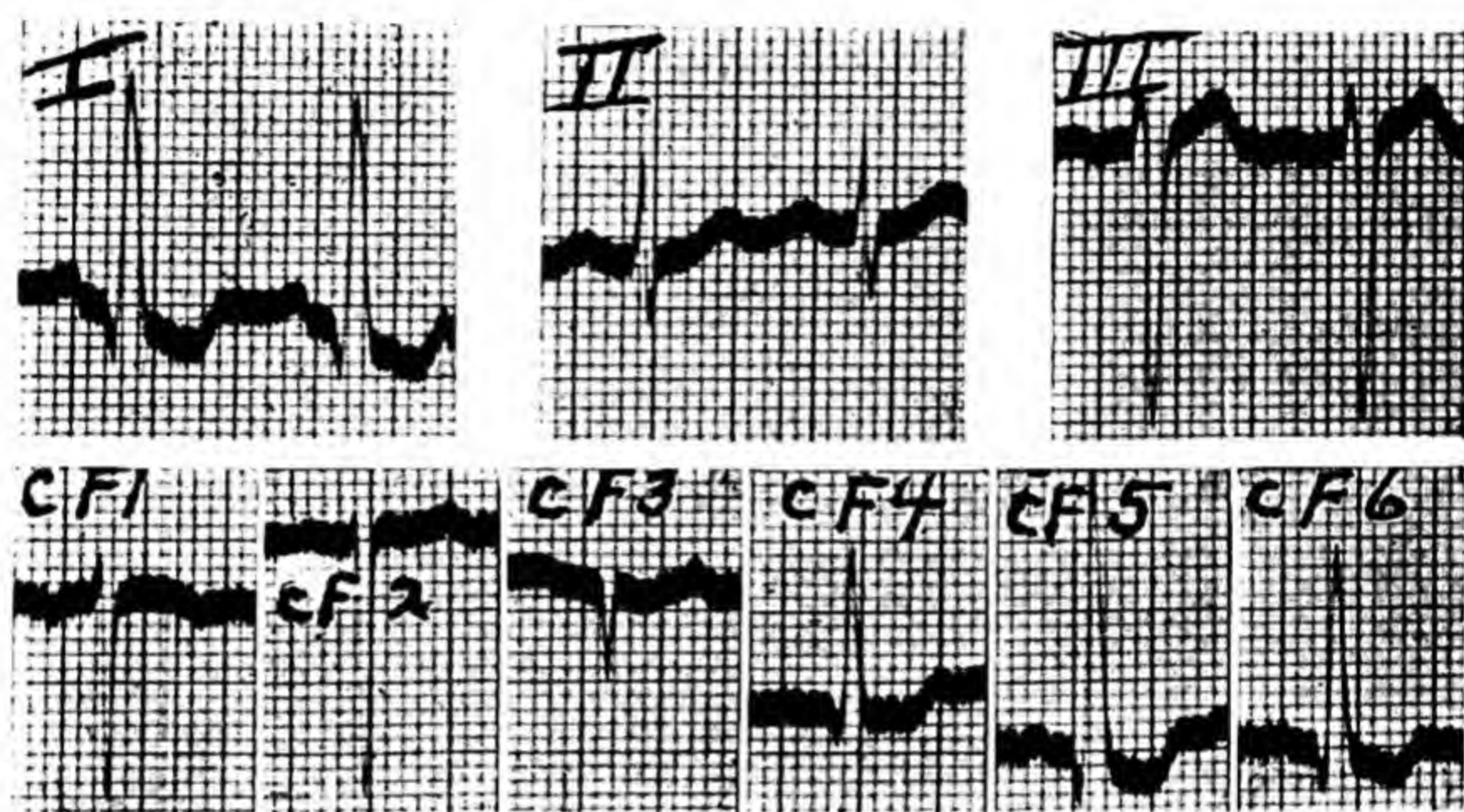


Fig. 92.—Intraventricular Block. The upper set shows three conventional leads. QRS = 0.1 second. The lower set taken from the six positions across the precordium (CF<sub>1</sub> to CF<sub>6</sub>) shows only very slight delay in onset of intrinsic deflection in positions CF<sub>4</sub> to CF<sub>6</sub>, thus ruling out left bundle branch block. The patient was a woman fifty-eight years old who was treated for pernicious anemia for twelve years and had angina for six years. Her blood pressure was 145 systolic and 85 diastolic. Examination of the heart revealed no abnormality.

curves will be found when there is little else to make one suspect heart disease and in that way they become very helpful diagnostically. In fact, the greatest value of electrocardiography is in the detection of significant abnormalities in the ventricular complexes when the rest of the examination reveals no essential abnormality.

### VENTRICULAR PREPONDERANCE (AXIS DEVIATION)

The height of the complexes in the various leads is determined by the electrical axis of the heart. Normally R waves are highest in Lead II and the sum of the waves in Leads I and III is approximately equal to Lead II. If the heart lies in an abnormal position, either pushed to one side or another by fluid in the chest, a high diaphragm or by malformation of the chest, it may be of normal structure and yet show different relationships in these complexes as a result of a shift in its axis. These changes are not great, to be sure, but need to be considered in interpreting electrocardio-



grams. In Figure 93 it will be seen that the R wave is highest in Lead I rather than in Lead II and yet the heart was normal. This was caused by a high diaphragm that is so common in obese individuals.

The electrical axis will also shift to one side or another if one ventricle enlarges more than the other. At birth the right ventricle is larger than the left and only after several months is the reversed relationship established. Contrariwise, during the normal aging process the left ventricle often becomes slightly hypertrophied even when, from a practical point of view, it is to be regarded as normal. The electrocardiogram will show

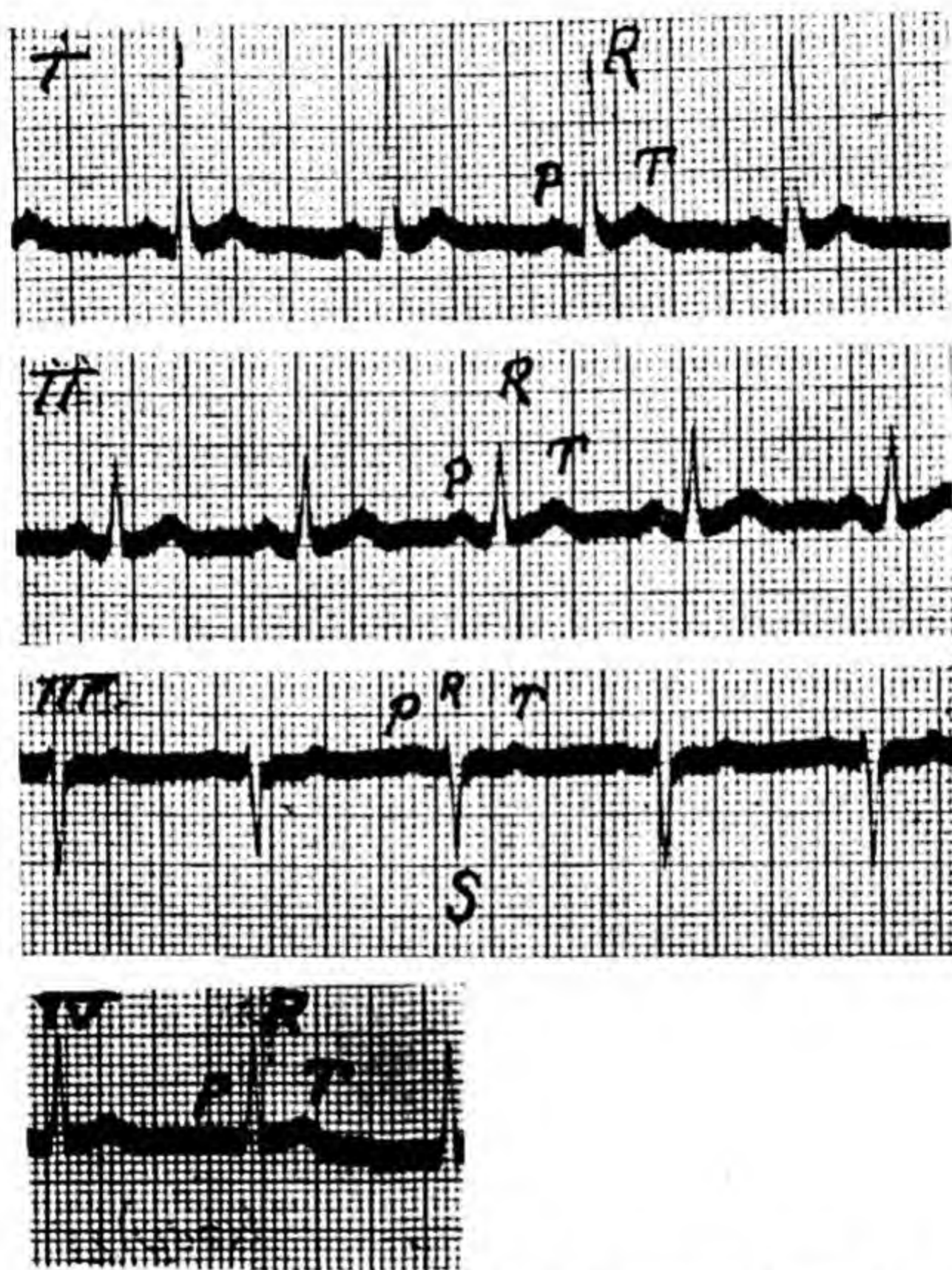


Fig. 93.—Left Axis Deviation. Note that the highest upward wave (R) of the initial ventricular deflection is in Lead I, and the lowest downward wave (S) is in Lead III. Lead IV is normal. This patient was a normal man, forty-one years old.

evidence of this undue preponderant hypertrophy of either ventricle. When the highest upward initial deflection (R wave) occurs in Lead I and the lowest downward deflection (S wave) in Lead III it denotes *left ventricular preponderance* or left axis deviation (Fig. 93). If the changes are reversed and the lowest downward wave is in Lead I and the highest upward deflection is in Lead III, *right ventricular preponderance* or right axis deviation is indicated (Figs. 94, 95). When both ventricles enlarge equally no change in the electrical axis results and the normal relations persist. With marked hypertrophy or dilation of the ventricles the QRS complex may be increased in duration to a slight extent. When this lasts



0.1 second or more it is most likely due to a delay or block in one of the branches of the bundle of His.

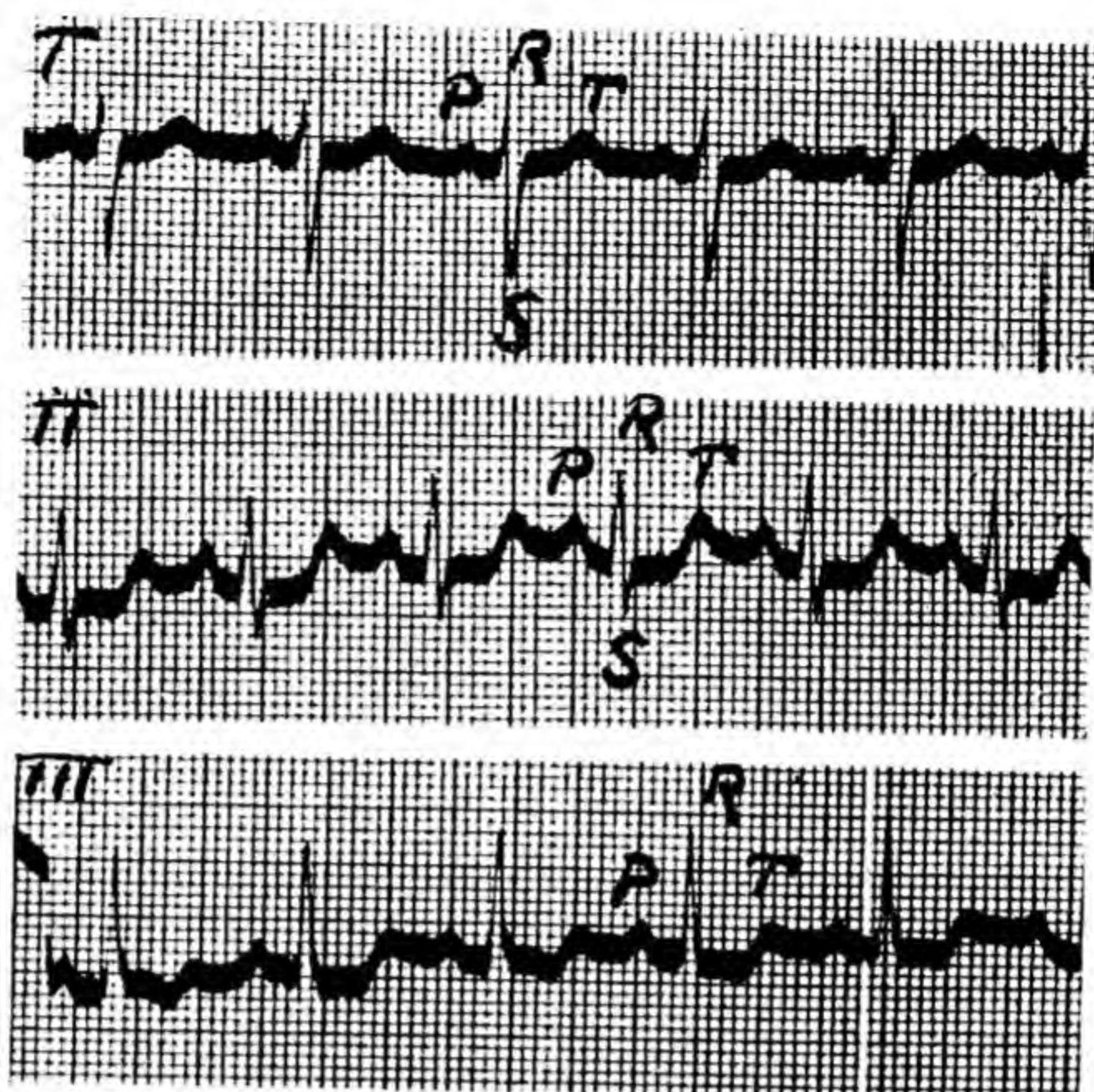


Fig. 94.—Right Axis Deviation. Note that the highest upward wave (R) of the initial ventricular deflection is in Lead III and the lowest downward wave (S) is in Lead I. This patient had mitral stenosis.

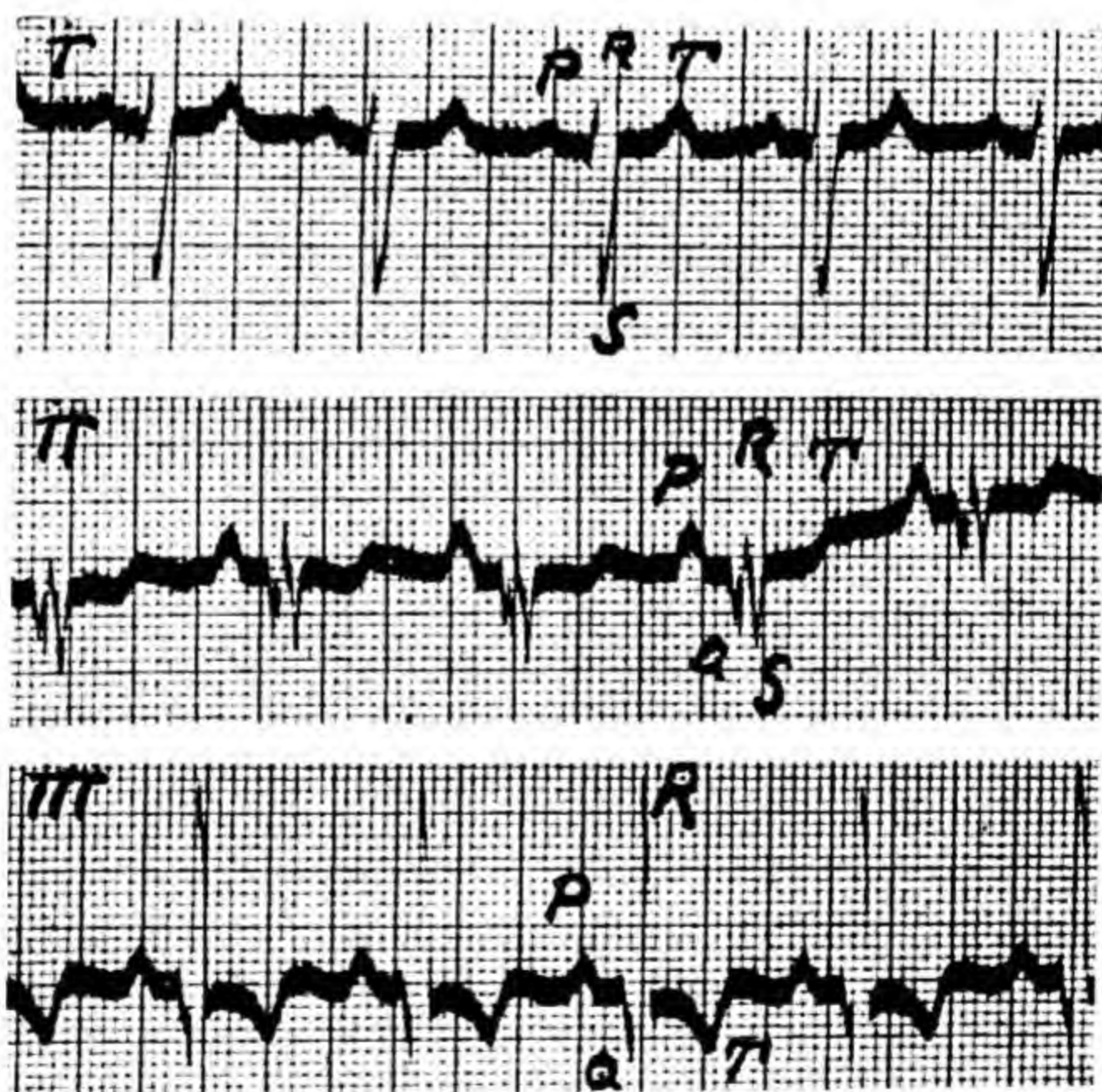


Fig. 95.—Right Axis Deviation. Note the deep S in Lead I and the high R in Lead III. These curves are somewhat similar to those in Fig. 94 but are more marked. Such extreme evidence of right ventricular preponderance generally occurs in congenital heart disease. This patient had pulmonary stenosis (tetralogy of Fallot).

Detection of ventricular preponderance or a shift of the electrical axis of the heart has its clinical significance. It has been mentioned that both right and left axis deviation may occur normally. In the interpretation of



electrocardiograms due regard must be given to these limitations. A heart must not be diagnosed as abnormal on these findings alone unless the changes are very marked. It is thought, however, that when left axis deviation is present and  $S_2$  is at least 25 per cent of the greatest R wave, organic heart disease usually exists. Evidence of left ventricular preponderance can often be made to disappear by having the patient take a deep breath (Fig. 96). With the descent of the diaphragm the deep  $S_3$  may diminish in size or even become an upright  $R_3$ . There are occasions, however, when the finding of preponderant hypertrophy of one side or the other aids considerably in differential diagnosis. I recall seeing a war veteran who was supposed to have aortic valvular disease because there was a systolic and diastolic murmur and a systolic thrill at the base of the heart. The electrocardiograms showed marked right ventricular preponderance. This led to an entirely different interpretation, because if hypertrophy develops in aortic valvular disease, it is expected to involve the left rather than the right ventricle. More careful examination, in-



Fig. 96.—Effect of Deep Breath on Left Axis Deviation. The patient was a woman, forty-five years old, with a normal heart. Note disappearance of  $S_2$  and an upright  $T_2$  with deep inspiration.

cluding  $x$ -ray of the heart, showed that the condition was some form of congenital heart disease which is often associated with right ventricular preponderance. Similarly, an elderly woman had congestive heart failure, hypertension and auricular fibrillation. The diagnosis was non-valvular heart disease because there was no diastolic murmur to be heard. The electrocardiograms showed well-marked right ventricular preponderance. This threw some doubt on the diagnosis, because left preponderance should have been found with a senile heart and hypertension. Mitral stenosis was suspected despite the absence of a diastolic murmur, for this could account for the right ventricular enlargement. An  $x$ -ray showed a prominent left auricle and later, on postmortem examination, a fish-mouth mitral stenosis was found.

Hypertrophy of the ventricles does not always develop according to expectations. Disease of the aortic valve and hypertension should produce their effects on the left ventricle by increasing its work. Likewise mitral stenosis, congenital disease of the pulmonary valve or pure emphysema should increase the burden on the right ventricle by increasing the pressure in the pulmonary system. Although these predicted results generally occur, there are exceptions that are difficult to explain. In cases in which the primary strain is on the left ventricle, prolonged passive congestion



in the lungs with increased pulmonary pressure often results in hypertrophy of the right ventricle. The whole heart in this way may be involved in the burden that one might have thought would be limited to one chamber. These variations limit the value of interpretations of preponderant hypertrophy. However, such data can, at times, be useful in directing attention at conditions that might otherwise be overlooked. It particularly is helpful in appraising valvular conditions, especially when more than one valve is involved. Let us assume that a patient has an obvious aortic valvular lesion and shows right preponderance. This would lead one to suspect the additional diagnosis of mitral stenosis because, although no preponderant hypertrophy of either ventricle may be present with pure aortic involvement, it will be extremely rare to find it associated with a shift of the axis to the right. Instances of very marked right preponderance almost always mean congenital heart disease (Fig. 95) and the extreme cases of left preponderance are not found except in some form of organic heart disease.

There are other changes in the ventricular complexes that need consideration in deciding whether the left ventricular preponderance may be regarded as normal or pathologic. In those cases in which the changes are of no clinical significance as far as myocardial disease is concerned,  $T_1$  will be upright,  $T_3$  inverted and there will be no  $S_2$ . There is some evidence to support the general idea that as one or more of those three factors change, the likelihood of myocardial disease increases, *i.e.*, a diminution in the height of  $T_1$ , the appearance of a well-defined  $S_2$  and an upright  $T_3$ . When left ventricular preponderance is associated with two or three of these changes one should hesitate in regarding it as insignificant.

### CONGENITAL HEART DISEASE

There are two types of electrocardiograms that are helpful in the diagnosis of congenital heart disease. The curves in true *congenital dextrocardia* are absolutely pathognomonic. The electrical axis of the heart lies in the direction opposite to normal in this condition, *i.e.*, from left to right. When the ordinary three leads are taken, therefore, Lead I will be a mirror picture of the normal Lead I. All the waves will be inverted (Fig. 97, upper tracing). Lead II will resemble a normal Lead III and *vice versa*. If the right and left arm electrodes are reversed and the leg electrode is placed on the right leg, curves of normal appearance will be obtained (Fig. 97, lower tracing). Care must be taken in making the diagnosis of dextrocardia from electrocardiograms, because accidental interchange of the arm electrodes will produce similar curves. This mistake is not at all uncommon.

The other type of electrocardiographic change that is found in congenital heart disease is marked right preponderance. This occurs particularly in cases of pulmonary stenosis and atrial septal defect but not in patent ductus arteriosus. The curves are often of larger amplitude than are seen in acquired valvular disease from which they need to be



distinguished. Conduction disturbances and biphasic initial ventricular complexes are fairly common in cases of ventricular septal defect. There are other abnormalities of the electrocardiograms with different congenital defects of the heart but, except for disturbances in rhythm like congenital heart block, they have not been sufficiently studied to have diagnostic value.

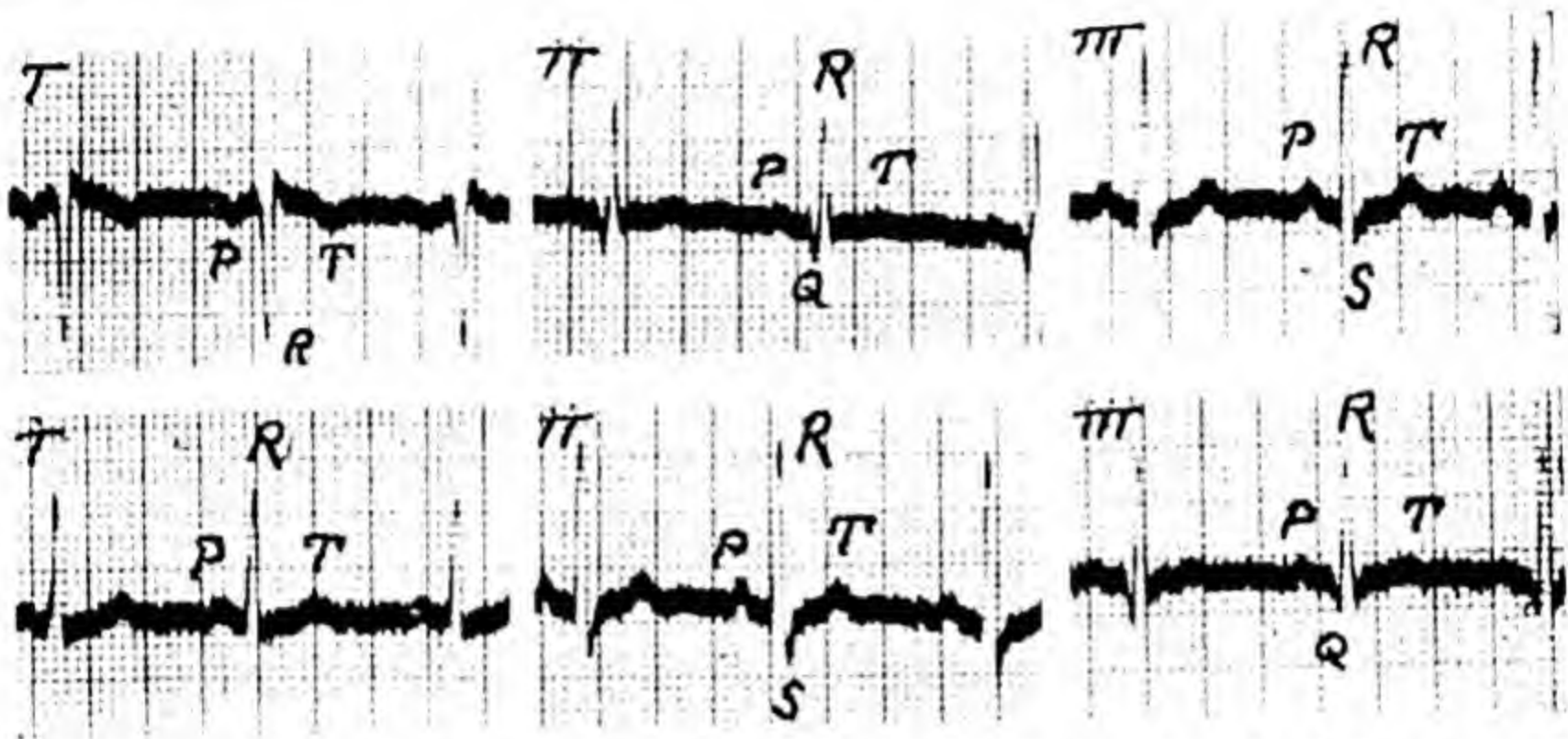


Fig 97.—Dextrocardia. The upper tracing shows the ordinary three leads in a case of congenital dextrocardia with situs inversus of the abdominal viscera. Note that all the waves (P-R-T) are inverted in Lead I. Leads II and III appear interchanged. The lower curves were obtained on the same patient by placing the right arm electrode on the left arm and *vice versa*. They then appear to be normal curves.

### CHANGES IN THE FORM OF THE AURICULAR COMPLEX

The auricular complex (P wave) varies in form somewhat in normal individuals. In general, with faster rates the P is larger and with slower rates it is smaller. There is one peculiar type of P wave that is present

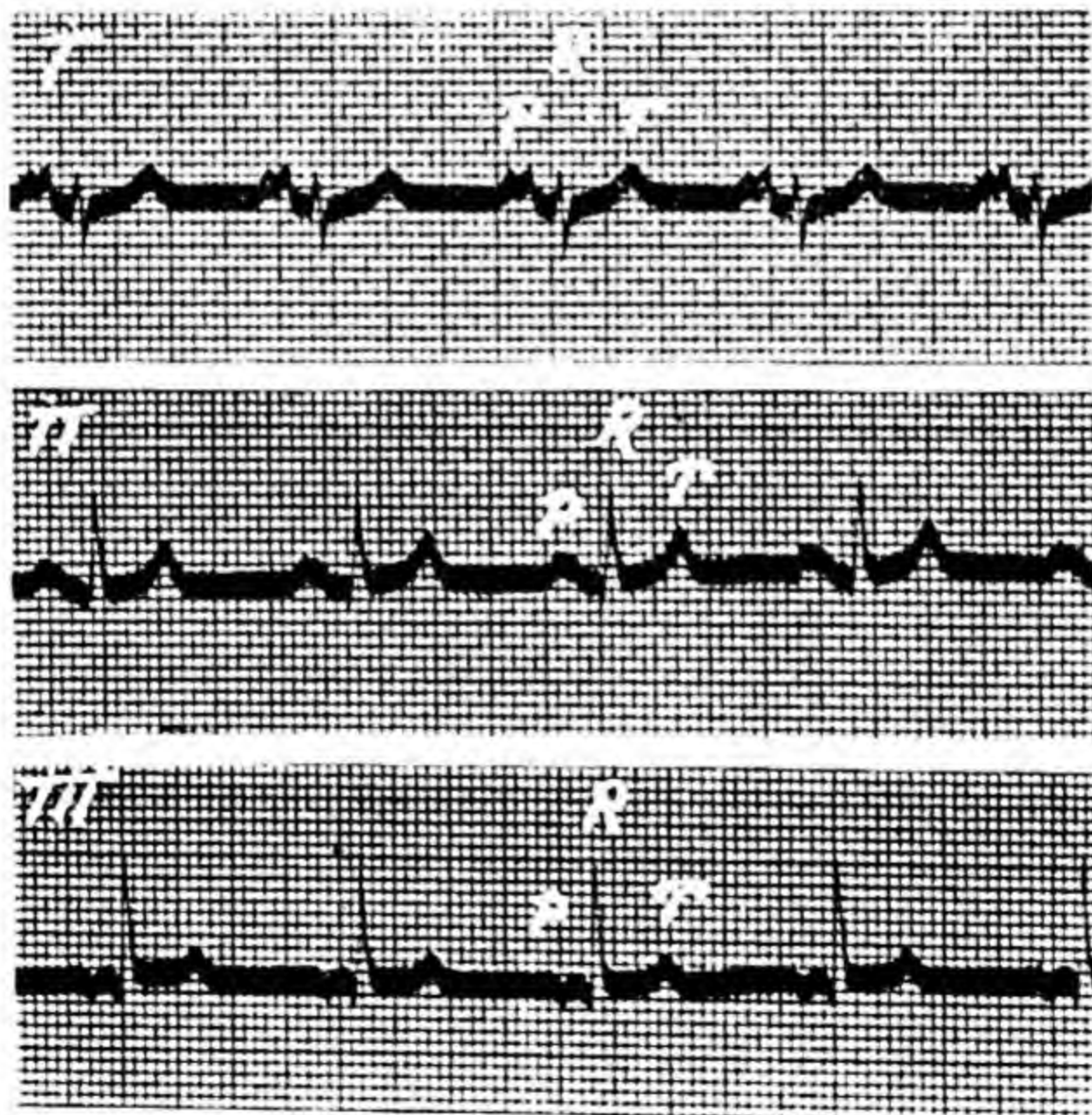


Fig. 98.—Auricular Hypertrophy. Note that the P waves in Lead I are broad, flat-topped and notched. The patient had well-marked mitral stenosis.



so frequently with mitral stenosis that it is fairly diagnostic. It is a wave that is prominent, broad, somewhat flat-topped and notched (Figs. 98, 99). These characteristics are generally displayed in Leads I and II, and often in Lead III the P wave is inverted. This type of P wave denotes an enlargement of the auricles and as this occurs almost exclusively in mitral stenosis it has important clinical value. When curves are found, such as are represented in Figures 98 and 99, one can be quite certain that mitral stenosis is present. There may be an additional tricuspid stenosis but this never occurs without the presence of mitral stenosis as well.

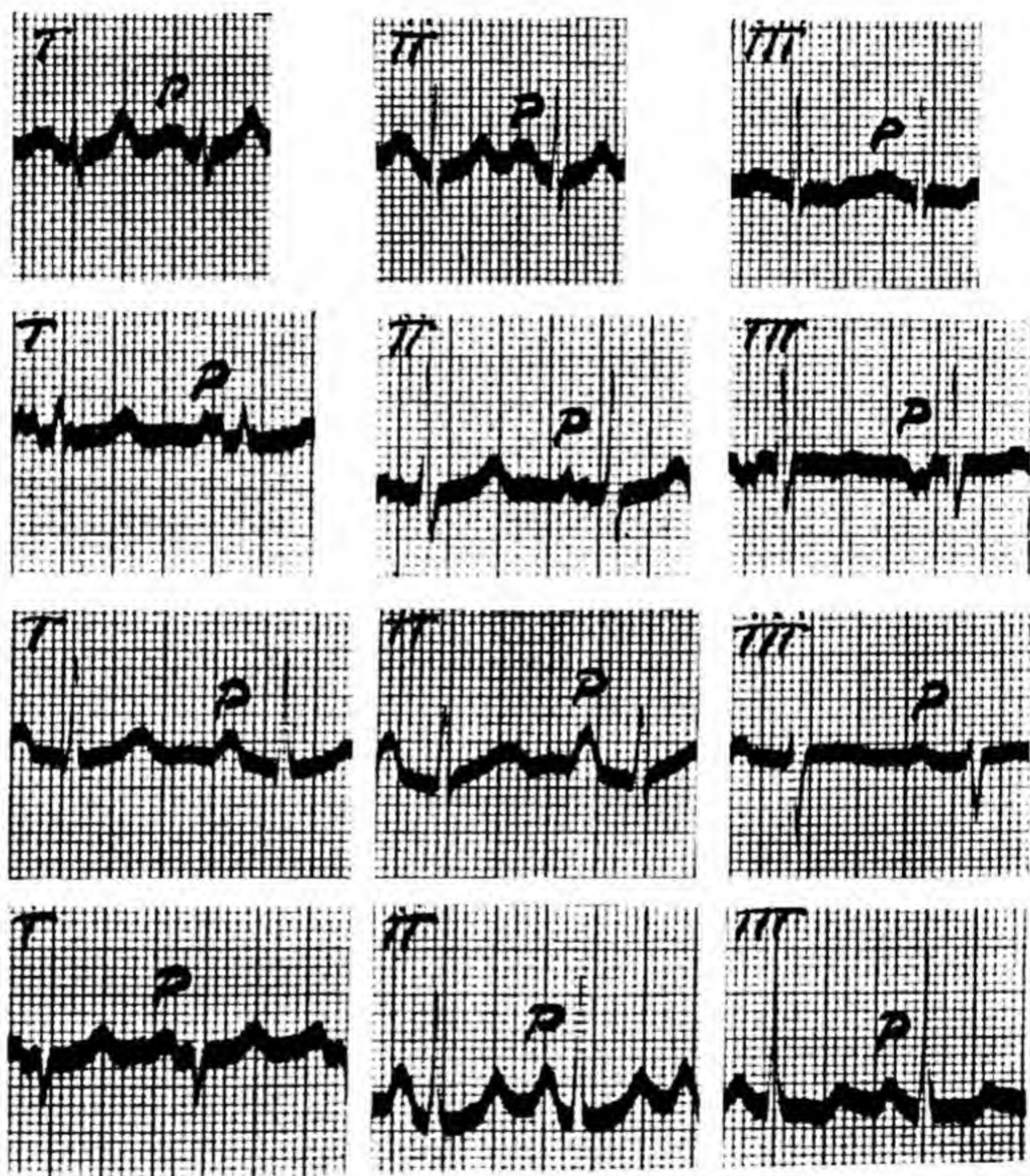


Fig. 99.—Auricular Hypertrophy. Four sets of curves showing large, broad and notched P waves. Note that P waves may be most prominent in Leads I or II and may be inverted in Lead III. All four patients had rheumatic mitral stenosis with or without aortic or tricuspid involvement.

Sharp, narrow and large P waves may be seen in hyperthyroidism and it is suspected that tricuspid stenosis may have a prominent P wave that is taller and narrower than those just described for mitral stenosis.

#### CHANGES IN FORM OF VENTRICULAR COMPLEXES

There now remains for consideration a heterogeneous group of conditions in which the ventricular complex becomes abnormal and the clinical significance of such changes. It will be seen that in some conditions the electrocardiographic findings are most important and helpful, in



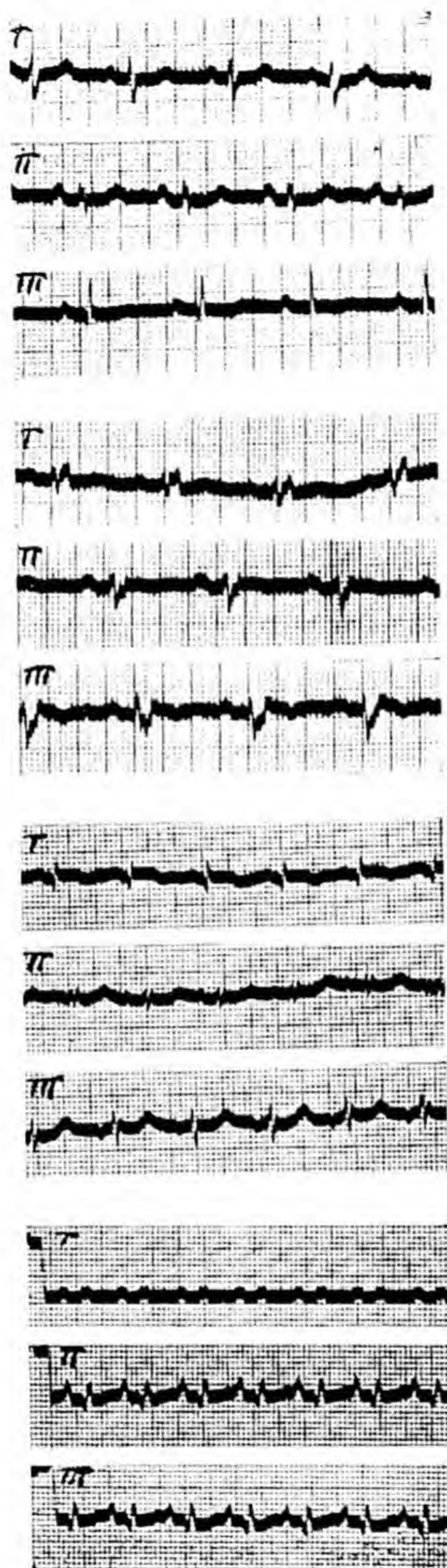


Fig. 100.—Abnormal Form of Ventricular Complex (Low Voltage). Four sets of curves showing QRS complexes of low amplitude in all leads. The lowest curves show the normal standardization. All four patients had serious heart disease of the coronary artery type.

others they may be only suggestive and frequently the changes may be misleading because they are not sufficiently distinctive.

**Ventricular Complexes of Low Amplitude.**—When the height of the R wave or the depth of the S wave is 5 mm. or less in all leads it is re-



garded as abnormal (Fig. 100, 101). Upward measurements should begin with the top of the iso-electric shadow of the string and those downward from the bottom of it. Such low curves may occur with advanced cardiac failure from any cause, either valvular or non-valvular, with acute coronary occlusion, anemia, myxedema, pericardial effusion or any condition showing considerable edema. They are also frequently present, even

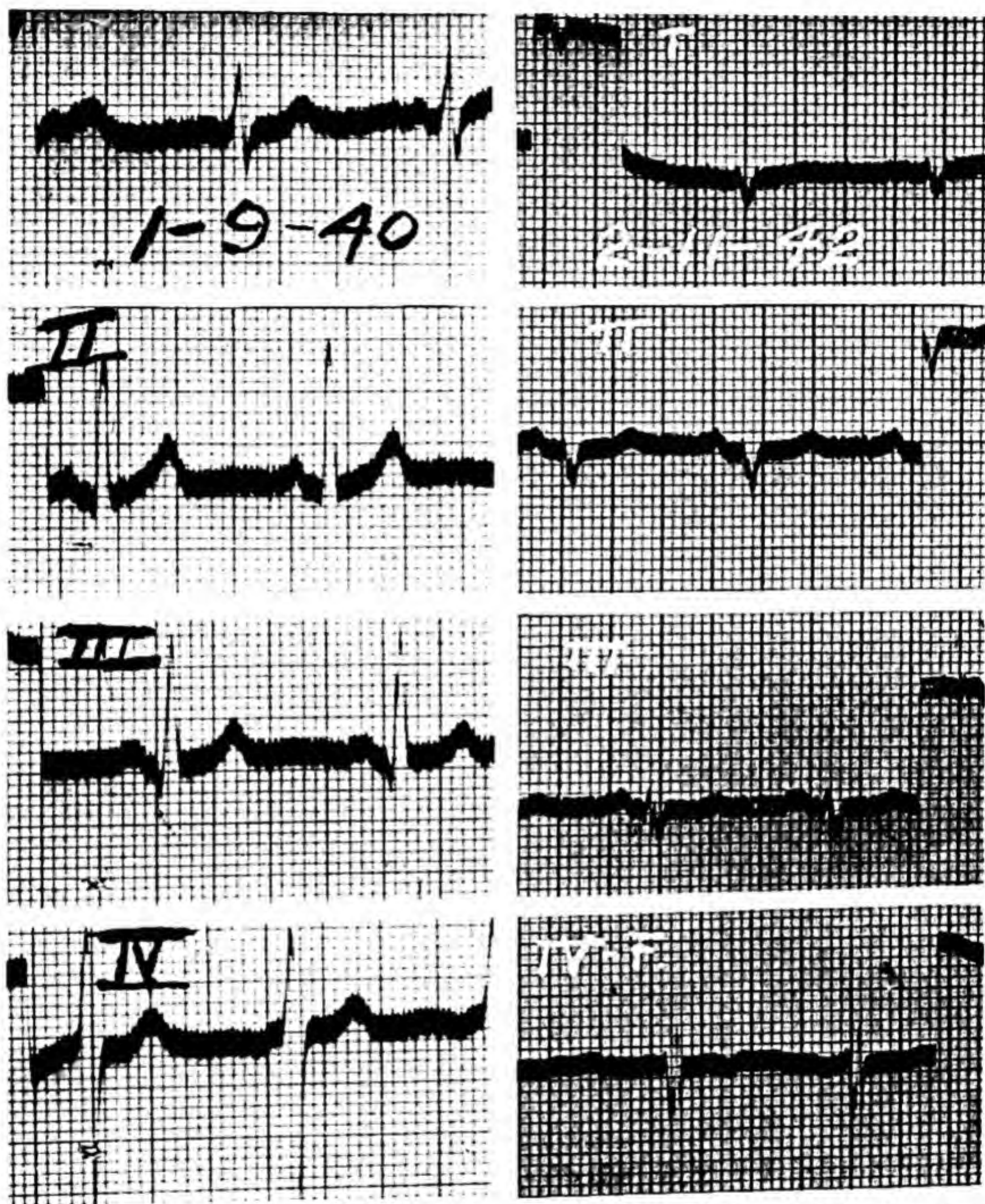


Fig. 101.—Abnormal Form of Ventricular Complex (Low Voltage). The first set, taken January 9, 1940, shows nothing very abnormal. An attack of acute coronary thrombosis occurred in the summer of 1940. The second set, taken February 2, 1942, shows marked decrease in amplitude of QRS and T waves. The patient had no peripheral edema, although there was a moderate right hydrothorax.

without evidence of heart failure, in patients with well-marked emphysema of the lungs. The low complexes are either due to the development of an electromotive force of small potential within the heart or to some electrical effect of the edema or tissues around the heart. The curves may become larger if the edema disappears or the congestive state improves. In myxedema (Fig. 102) not only are the QRS waves dimin-



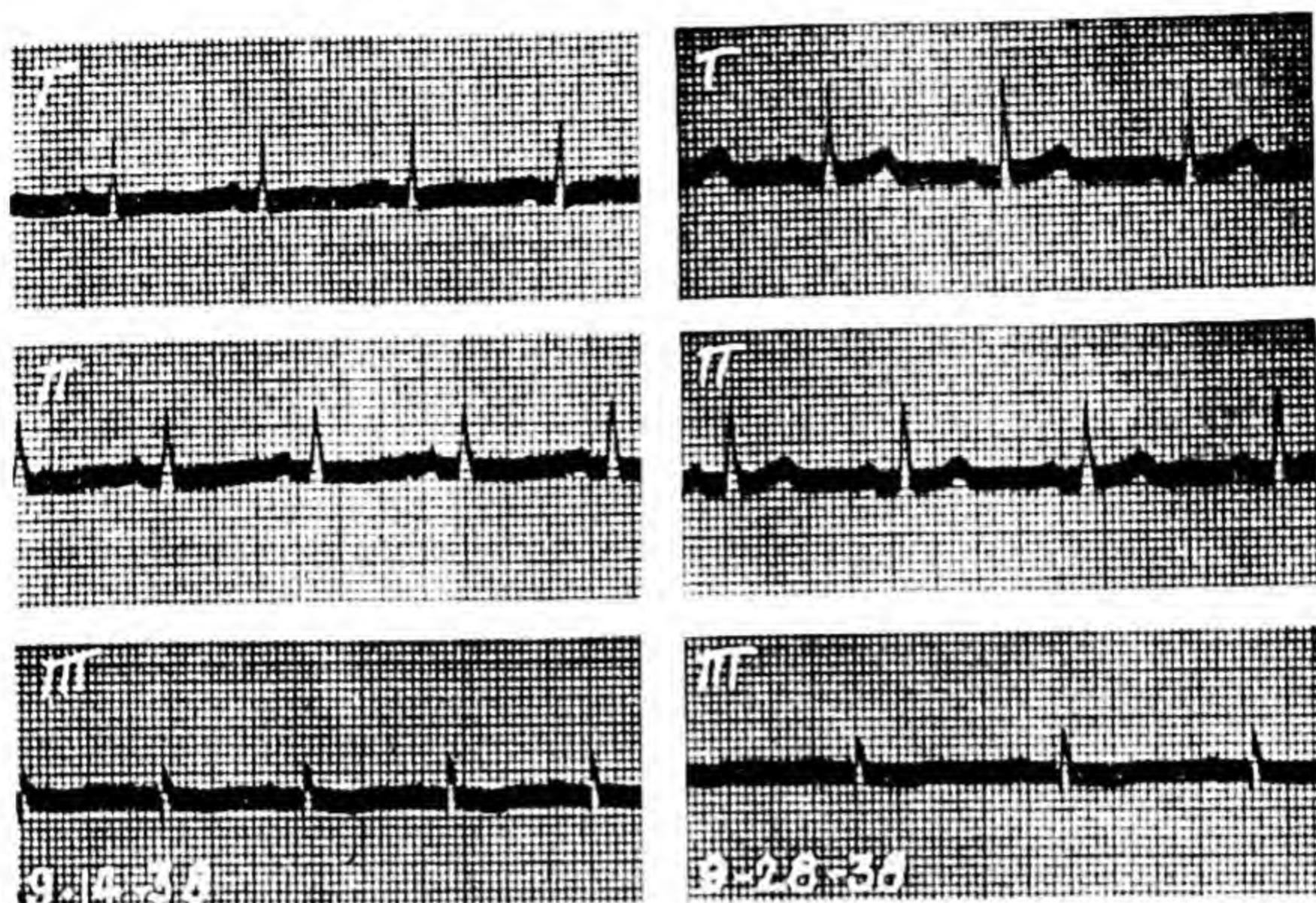


Fig. 102.—Effect of Myxedema. The first set was taken September 14, 1938 when the basal metabolic rate was  $-35$  per cent. The second set taken fourteen days later, after thyroid therapy, the basal metabolic rate being  $-10$  per cent. Note the increase in height of T and R waves.

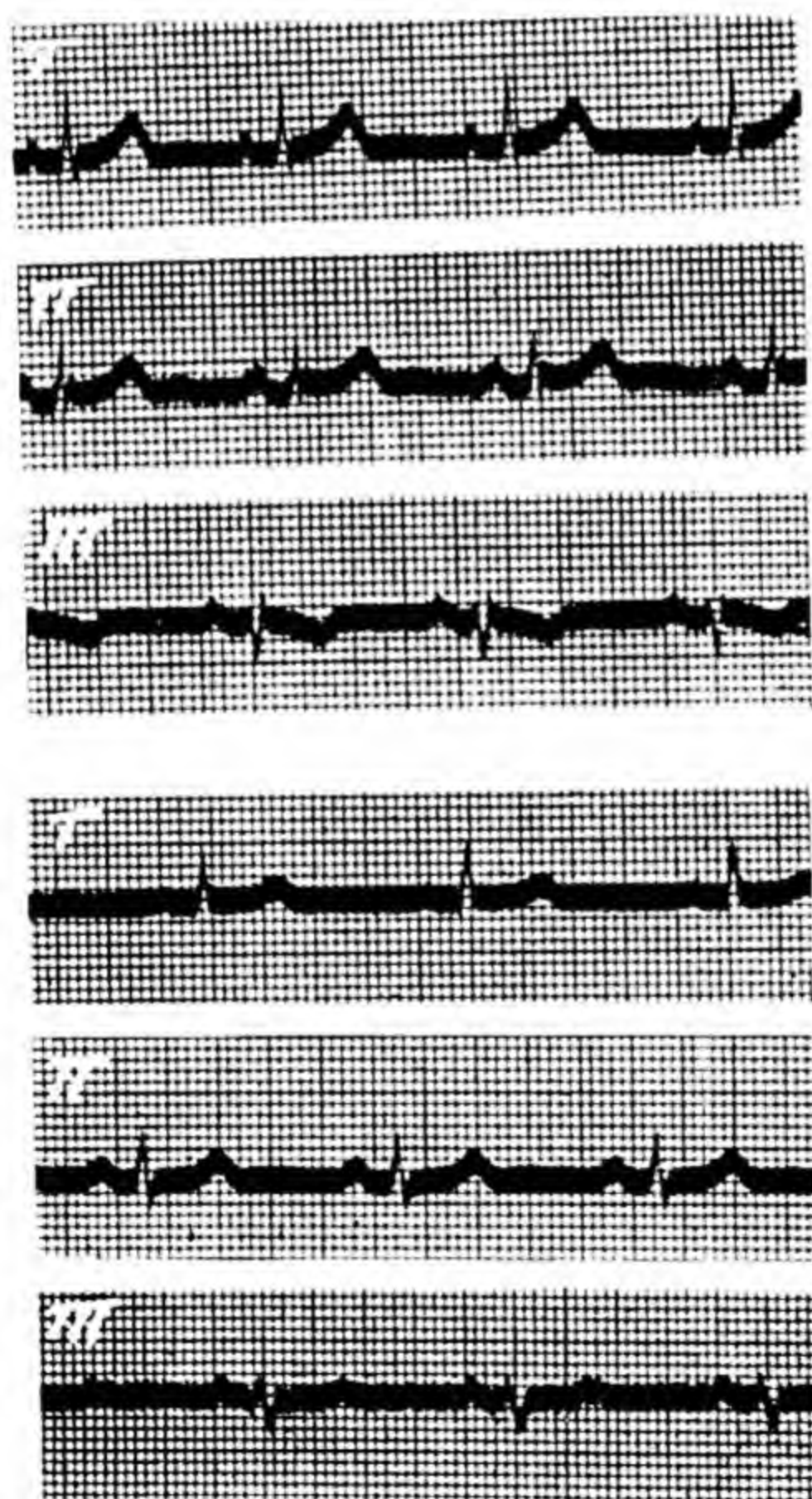


Fig. 103.—Low Voltage. Two sets of curves showing QRS waves of low amplitude. Note that the general form of the ventricular complexes is normal (upright  $T_1$  and  $T_2$  and sharp R waves) in contrast to those in Fig 100. These two patients had no heart disease.



ished, but the T waves are flat. Such curves can return to normal on thyroid therapy. When they are found after a coronary occlusion they are apt to persist indefinitely and be compatible with a satisfactory state

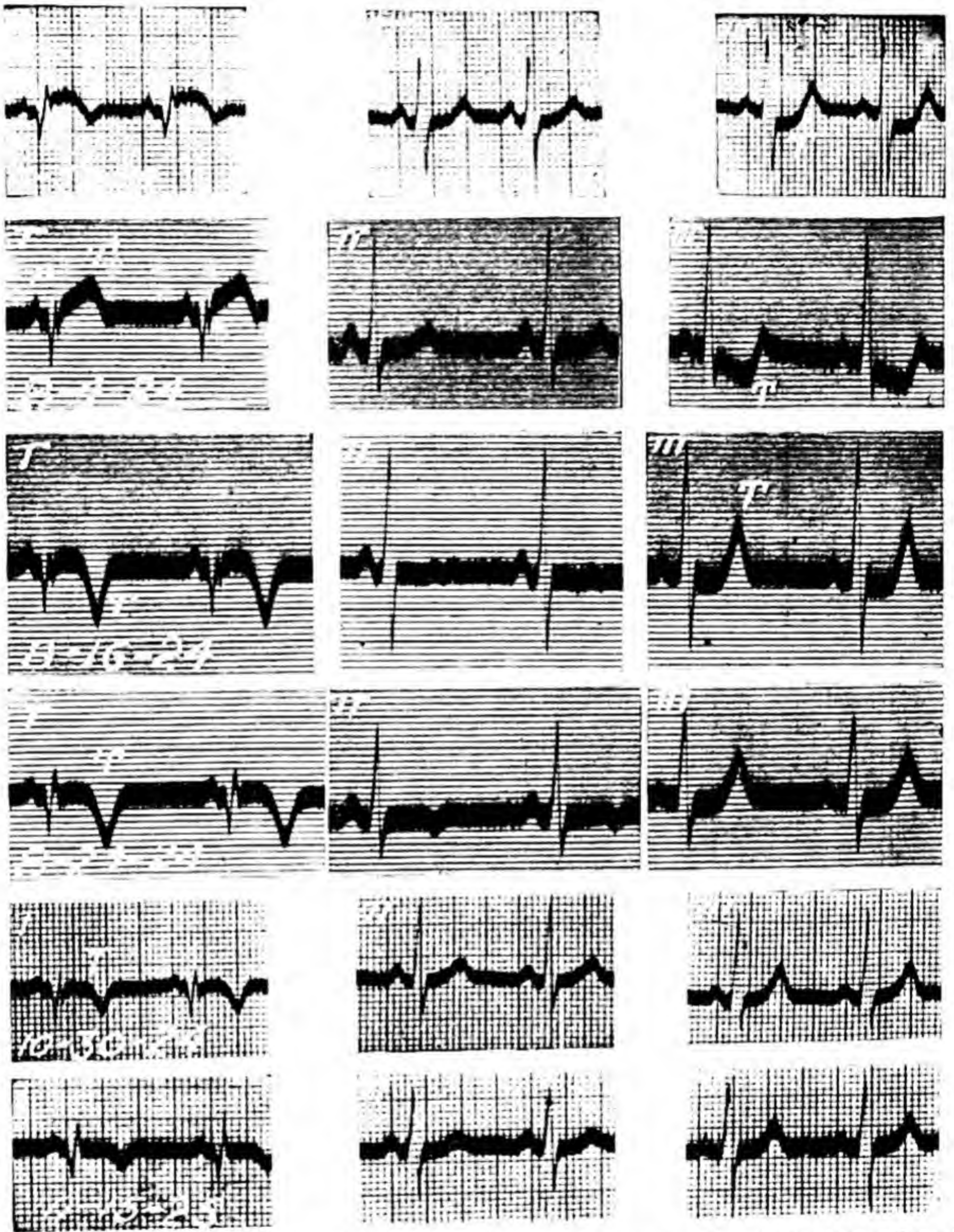


Fig. 104.—Abnormal Form of Ventricular Complex (Coronary Type). A series of tracings following an attack of acute coronary thrombosis which occurred July 20, 1924. Note that the high take-off of the T wave and the subsequent sharp inversion occur in Lead I. Somewhat similar changes but in the opposite direction occur in  $T_1$ . Also there is a distinct  $Q_1$  which persists even the following year. These curves represent infarction of the anterior part of the ventricle. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

of health for years. In general, curves of very low amplitude indicate a fairly grave condition of the heart, but there are exceptions to this rule.

There are tracings that show ventricular complexes of low amplitude that require a different interpretation. They are not apt to be quite as small, although they may be, but they appear otherwise normal (Fig.



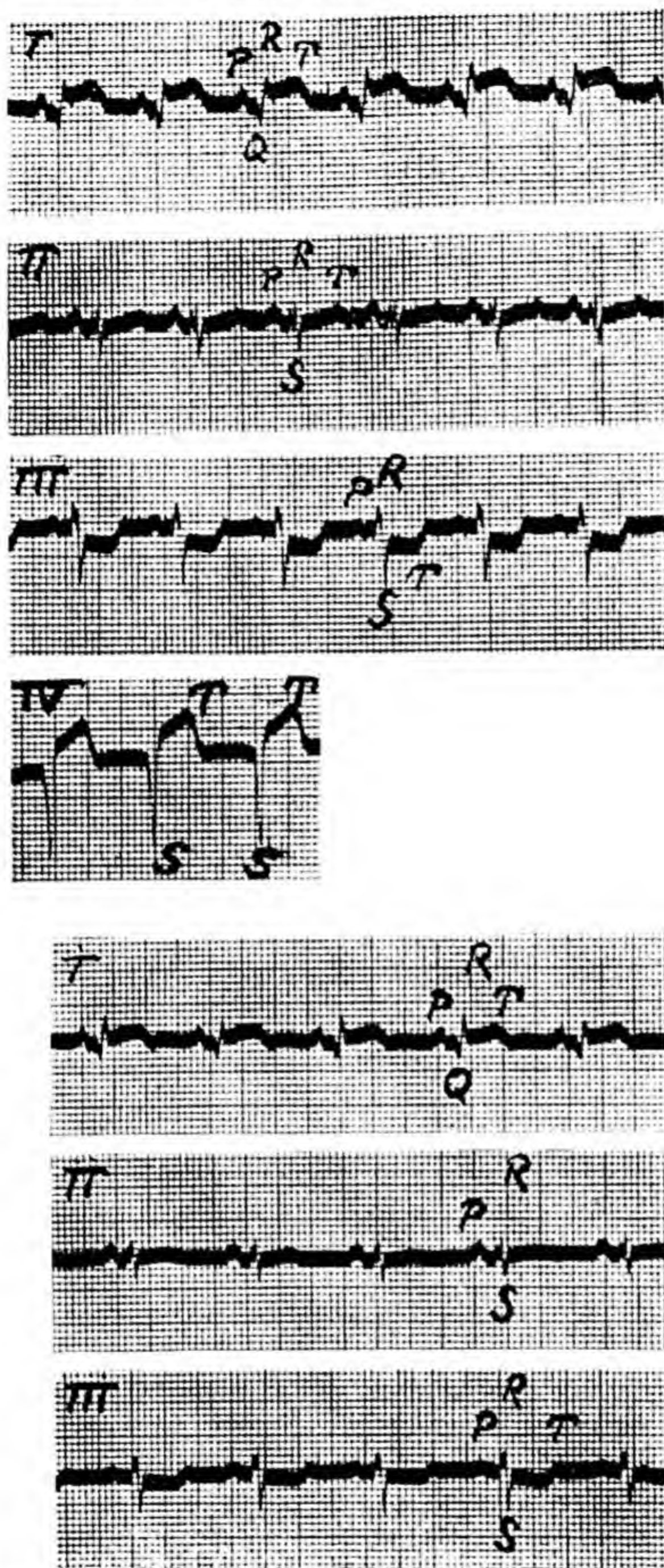


Fig. 105.—Abnormal Form of Ventricular Complex (Coronary Type). The upper curves were taken June 5, 1935; the lower, June 10, 1935. Note the high take-off of the R-T interval in Lead I and depressed interval in Lead III. A small  $Q_1$  is present. Lead IV shows complete absence of initial upward deflection ( $R_4$ ) and an elevated S-T segment. In the lower three leads the changes are less prominent. These curves are typical of infarction of the anterior part of the left ventricle. The patient had marked weakness and sweating with but very little discomfort in his chest and the case was first diagnosed as fever, unknown origin, before these curves were obtained.

103) The QRS waves are not notched or spread and the T waves have a normal configuration. These points distinguish them from the abnormal ones described above. They are found in individuals who are well



and show no evidence of organic heart disease. It is evident, therefore, that ventricular complexes of low potential must be interpreted with due care and should be used only in conjunction with other data as indicative of heart disease.

**Ventricular Complexes in Coronary Thrombosis and Myocardial Infarction.**—The most important contribution that electrocardiography

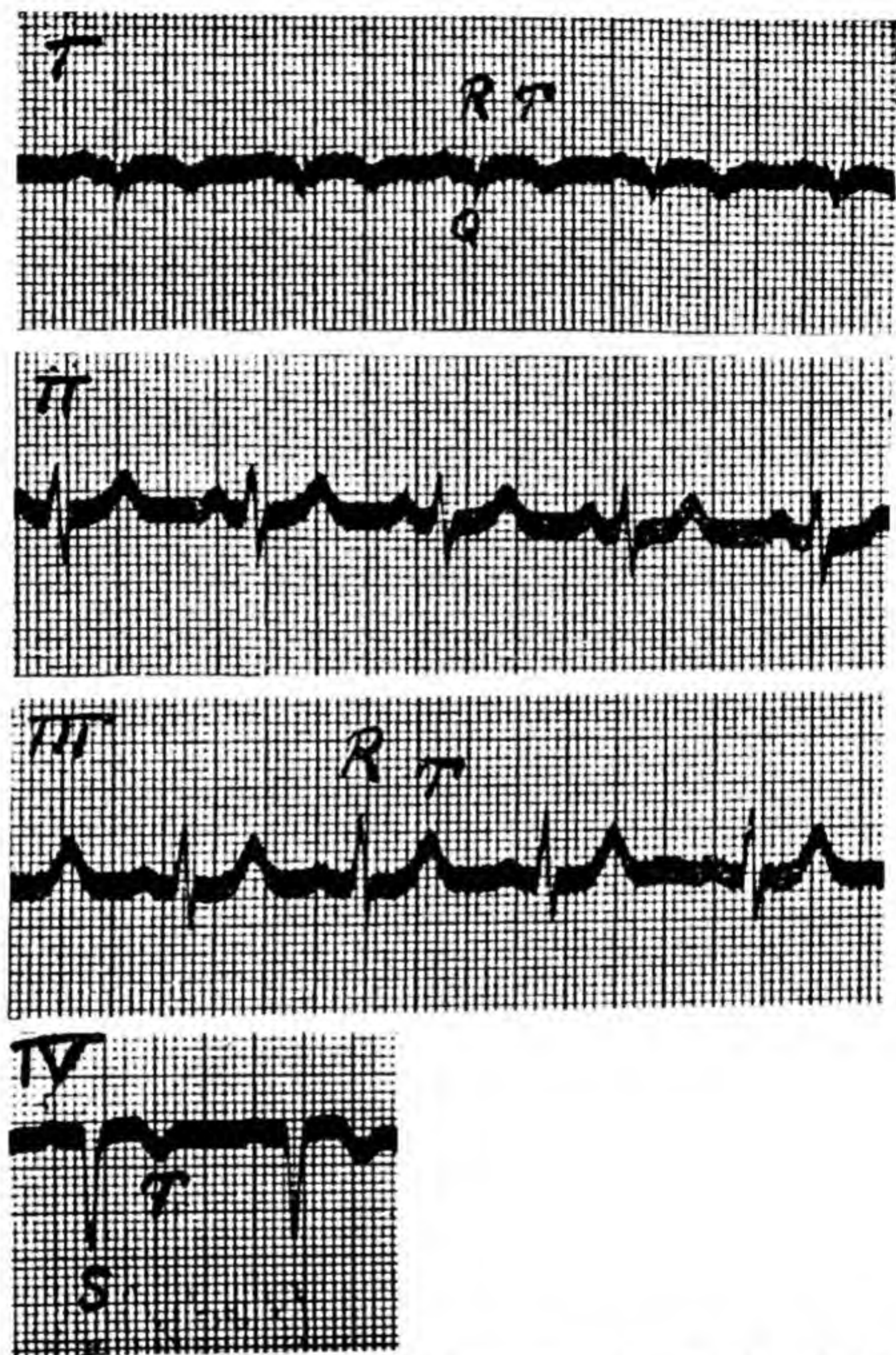


Fig. 106.—Abnormal Form of Ventricular Complex (Coronary Type). Note a small  $Q_1$  slightly rounded and dipped R-T interval in Lead I and a high upright  $T_1$ . These three conventional leads are extremely suggestive of a coronary thrombosis with an anterior lesion but the absence of  $R_4$  and inverted  $T_4$  from the apex are conclusive. The attack occurred four months before these electrocardiograms were made.

has made to medicine is in the diagnosis of coronary thrombosis and myocardial infarction. This goes back to the early observations of Pardee when he first called our attention to the displacement of the S-T segment in acute myocardial infarction. When the ventricular complexes change after such an attack they do so because of alterations in the musculature supplied by the vessel involved and not because of the thrombosis itself. It would be more proper to designate these abnormalities



as indicative of myocardial infarction, which generally results from coronary thrombosis but may come from disease of the arteries such as narrowing without thrombosis.

Anterior Infarction.—When an acute occlusion of a main coronary artery occurs there follows a sequence of striking abnormalities in the ventricular complexes. The degree and character of such changes vary

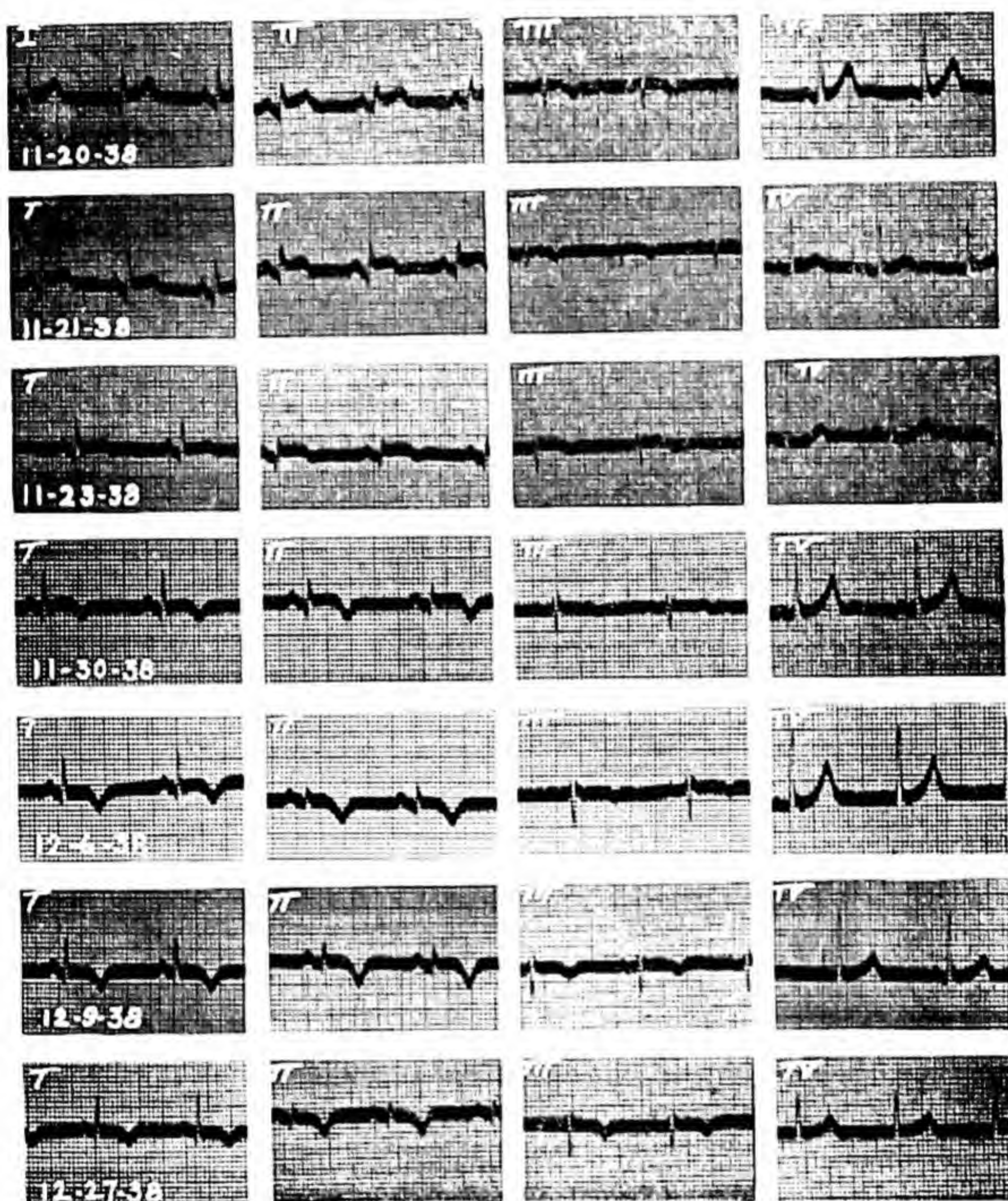


Fig. 107.—Coronary Type of Ventricular Complex. The first set resembled curves seen in rheumatic carditis. Subsequent changes in R-T segment with final rounding and dipping pointed to myocardial infarction. Note significant changes in customary three leads while Lead IV remains essentially normal. It is unusual that the T wave becomes inverted in all three leads.

considerably in different cases and depend, among other factors, upon the location of the artery involved (anterior or posterior), the extent of muscle infarction and probably on whether the lesion reaches the endocardium or pericardium.

Figure 104 represents a typical series of curves in a case in which there was thrombosis of the descending branch of the left anterior coronary



artery with intarction of the anterior and apical portions of the left ventricle. Figures 105 to 110 are illustrative of similar cases. The first changes to take place are an elevation of the R-T segment in Lead I (high take-off) and a depression in Lead III (low take-off). These may be noted as early as a few hours or possibly minutes after an acute attack, although they may not take place for a day or two. During the subsequent days the R-T segment undergoes further changes. The elevation becomes less and less but the interval tends to retain a curved form with the convexity upward (see Fig. 108, Lead I). The T wave gradually becomes inverted and sharply dipped. While these changes are going on

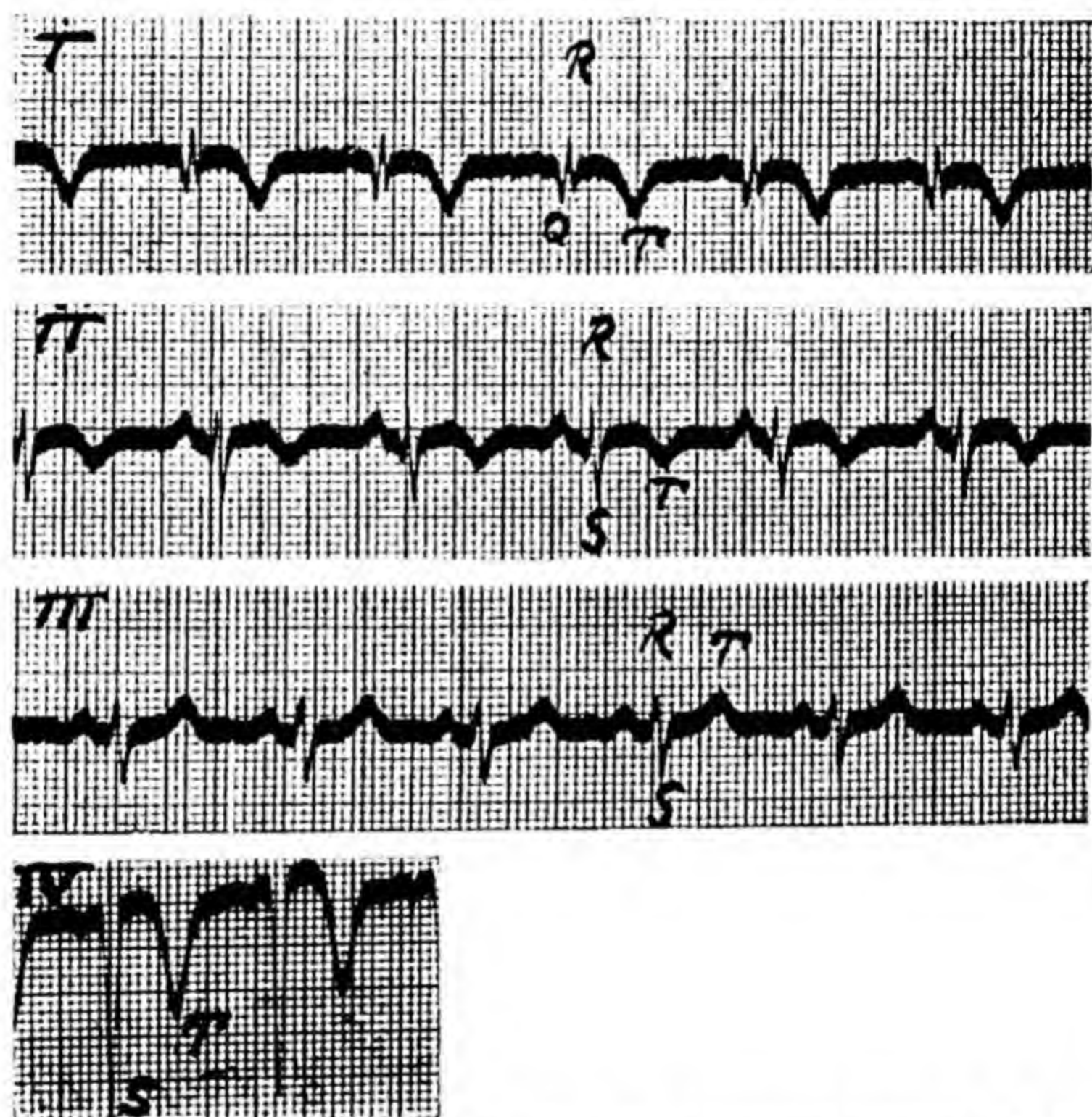


Fig. 108.—Coronary Type of Ventricular Complex (Anterior Lesion). Note the rounded and sharply inverted  $T_1$  and  $T_2$  and a small  $Q_1$ . Lead IV shows no  $R_4$  and a deep inverted  $T_4$ . (Normally there should be an R and upright T.) The attack was considered as due to "gastritis" three weeks before these tracings were made, because of vomiting for twenty-four hours and distress over the ensiform.

in Lead I the opposite is taking place in Lead III for there the T wave gradually becomes more and more upright (Fig. 104). These T waves may attain great size. Changes in the R-T portion of the ventricular complex, although often very marked, may be transient so that eventually even the normal form can be resumed. There may also be alterations in the initial ventricular complexes which generally become permanent. Lead I may show a Q wave which will persist for years (Fig. 104, lowest curve). In this way the permanent effects enable one to suspect that a coronary attack occurred in the past even when the T wave shows no definite abnormalities.



*Posterior Infarction.*—Infarction of the posterior or diaphragmatic portion of the left ventricle produces changes similar to those seen in anterior lesions, but they occur in different leads (Figs. 111 to 115). In the former the elevation of the R-T segment occurs in Lead III and often Lead II as well, while depression of the same segment is taking place in Lead I. The high take-off in Lead III gradually declines and there appears a convexed rounding and dipping of  $T_3$ . Finally  $T_3$  may become sharply inverted with a V-shaped configuration. While these changes are going on in Lead III, reciprocal alterations may be going on in Lead I. Like-

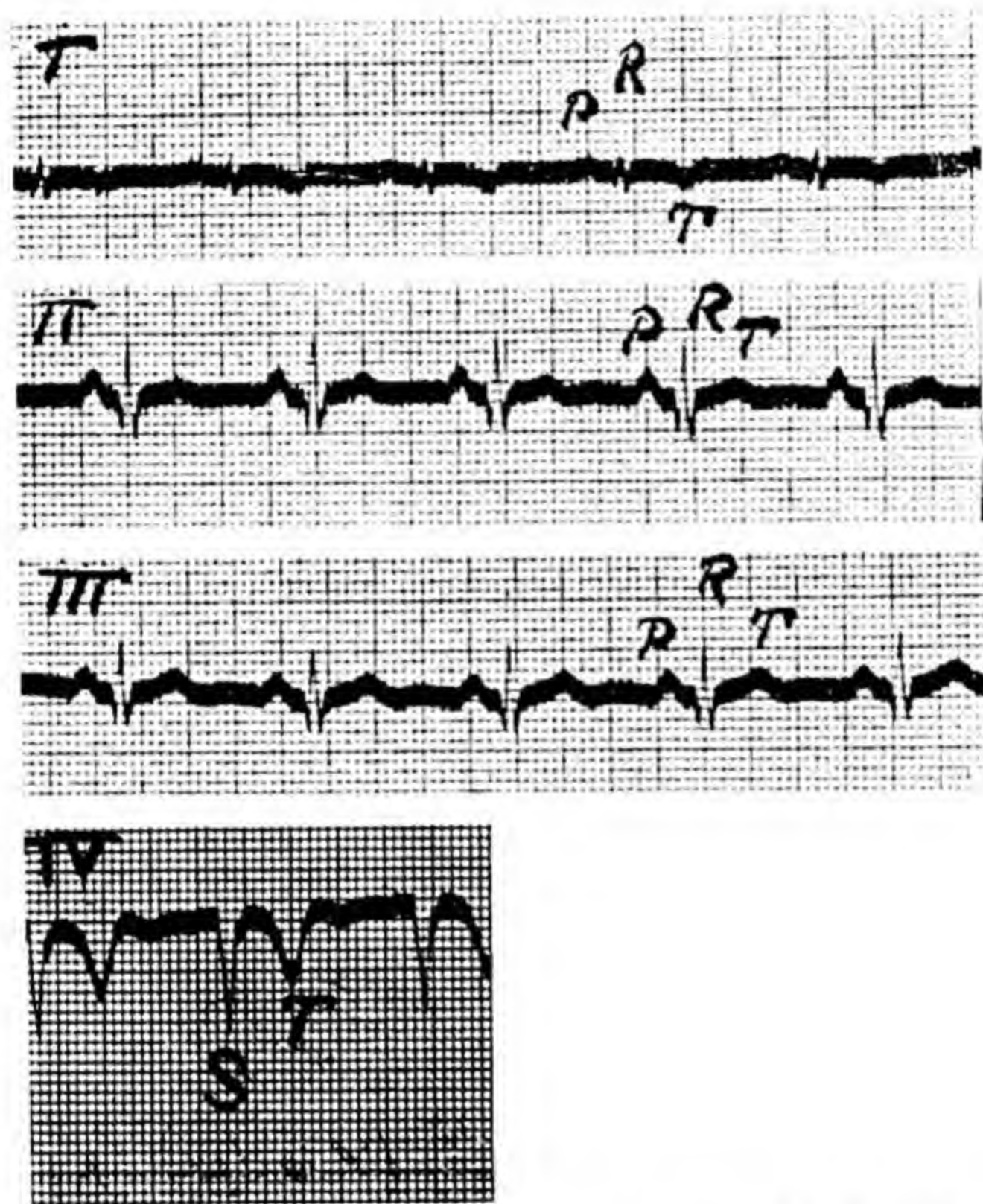


Fig. 109.—Coronary Type of Ventricular Complex (Anterior Lesion). Note the very slight rounding of the R-T segment in Lead I. This is barely suggestive of myocardial infarction, but the absent  $R_4$  and negative  $T_4$  are confirmatory of this diagnosis. The patient had a duodenal ulcer and came to the gastro-intestinal clinic because of a squeezing distress over the lower sternum.

wise a  $Q_3$  and at times a  $Q_2$  often develop in posterior lesions, just as a  $Q_1$  does in anterior lesions. The changes in the Q waves are likely to be permanent while those in the T waves are often transient. Figure 111 illustrates in a striking fashion the high take-off of  $T_2$  and  $T_3$  and the gradual marked inversion of these waves. It also shows the persistence of  $Q_3$  more than two years after the attack. A similar series of curves is shown in Figure 112. The first set of curves was obtained four hours after the onset of the attack and already the R-T interval is markedly elevated.



In interpreting the significance of the form of the R-T segment or of the T wave in all electrocardiographic work, the exact shape of the waves must be carefully observed. One should contrast the inverted T waves in Leads I and II of Figure 108 with those in the two middle sets of curves in Figure 133. The former are convexed upwards and have a terminal sharp dip and the latter are cup-shaped and concave. The one indicates myocardial infarction while the other denotes a digitalis effect.

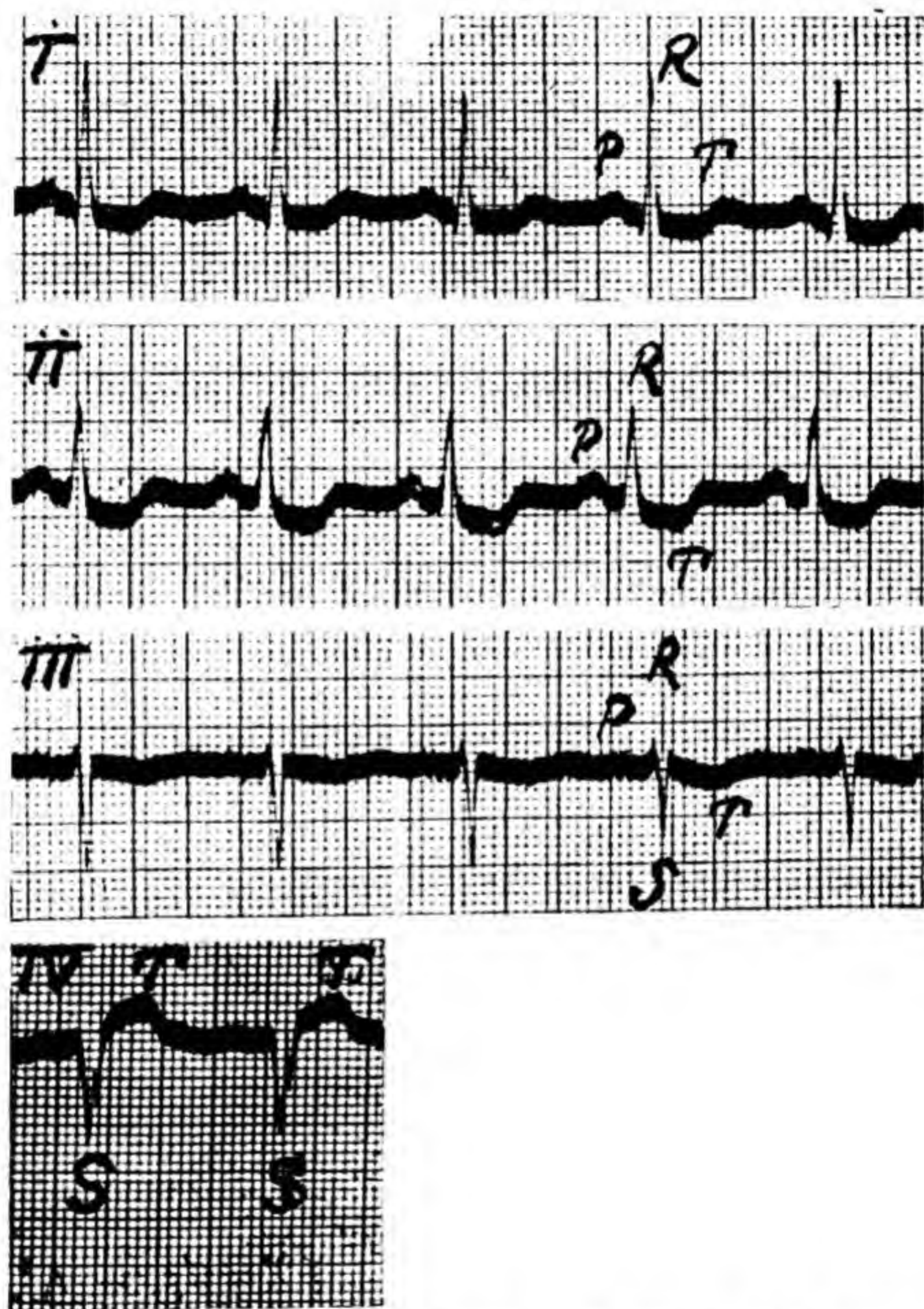


Fig. 110.—Coronary Type of Ventricular Complex (Lead IV). The customary three leads merely show slightly inverted T waves in all leads possibly due to digitalis. The absent R<sub>4</sub>, however, is reliable evidence of an anterior infarction of the ventricle. The patient had typical angina pectoris but no clinical history of an acute coronary thrombosis.

Various other combinations of electrocardiographic abnormalities occur in cases of coronary artery disease with myocardial infarction. Some are due to the development of a fresh lesion when an old infarct is also present. Curves may then have some features pointing to anterior and others to posterior involvement. It has been suggested by Wolferth that acute lateral lesions of the left ventricle will show depressed R-T in Lead IV, and usually in Leads I and II, absence of signs of posterior



infarction in Lead III, and usually no significant change in the QRS complex.

Electrocardiographic evidence of coronary artery disease may be uncovered, by taking tracings directly after effort (Fig. 116) or while the patient is inhaling 10 per cent oxygen. Marked deformity of the ventricular complex, particularly depression or elevation of the S-T segment, may be produced by these methods and will prove very helpful diagnostically.

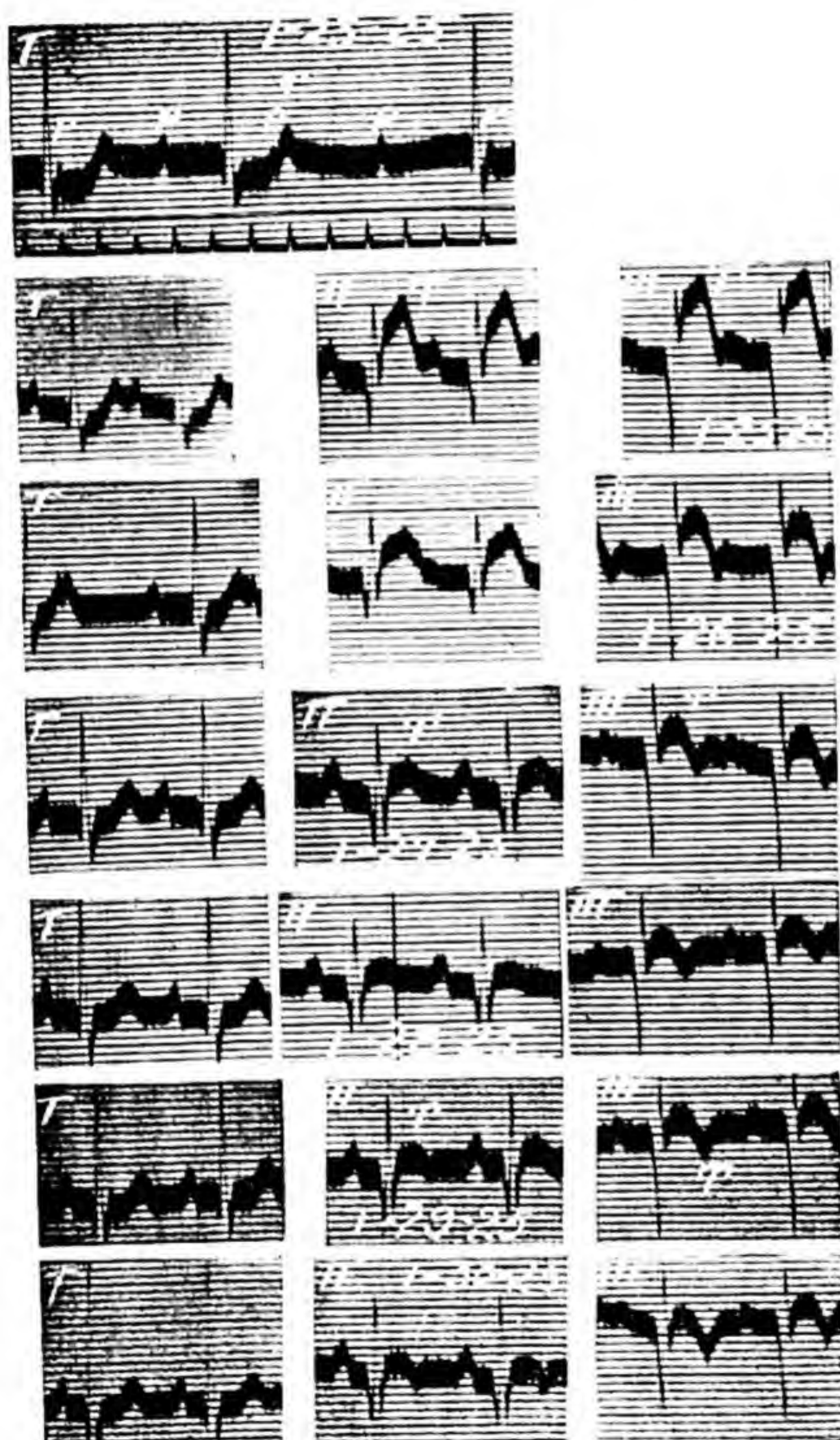


Fig. 111.—(Continued on page 392.)

*Precordial Lead (Lead IV).*—The introduction of *precordial* or *chest leads* (Lead IV) has thrown further light on the diagnosis of myocardial infarction. Earlier in this chapter the technique of this procedure was discussed, and mention was made of a change in terminology that has come into general use. The new recommendations of the American Heart Association will therefore be followed. The polarity is now such



that tracings in Lead IV obtained with the present technique are essentially a mirror picture of those formerly taken and of those published in the first edition of this book. It must be emphasized that whereas several precordial leads from different points near the heart may be necessary

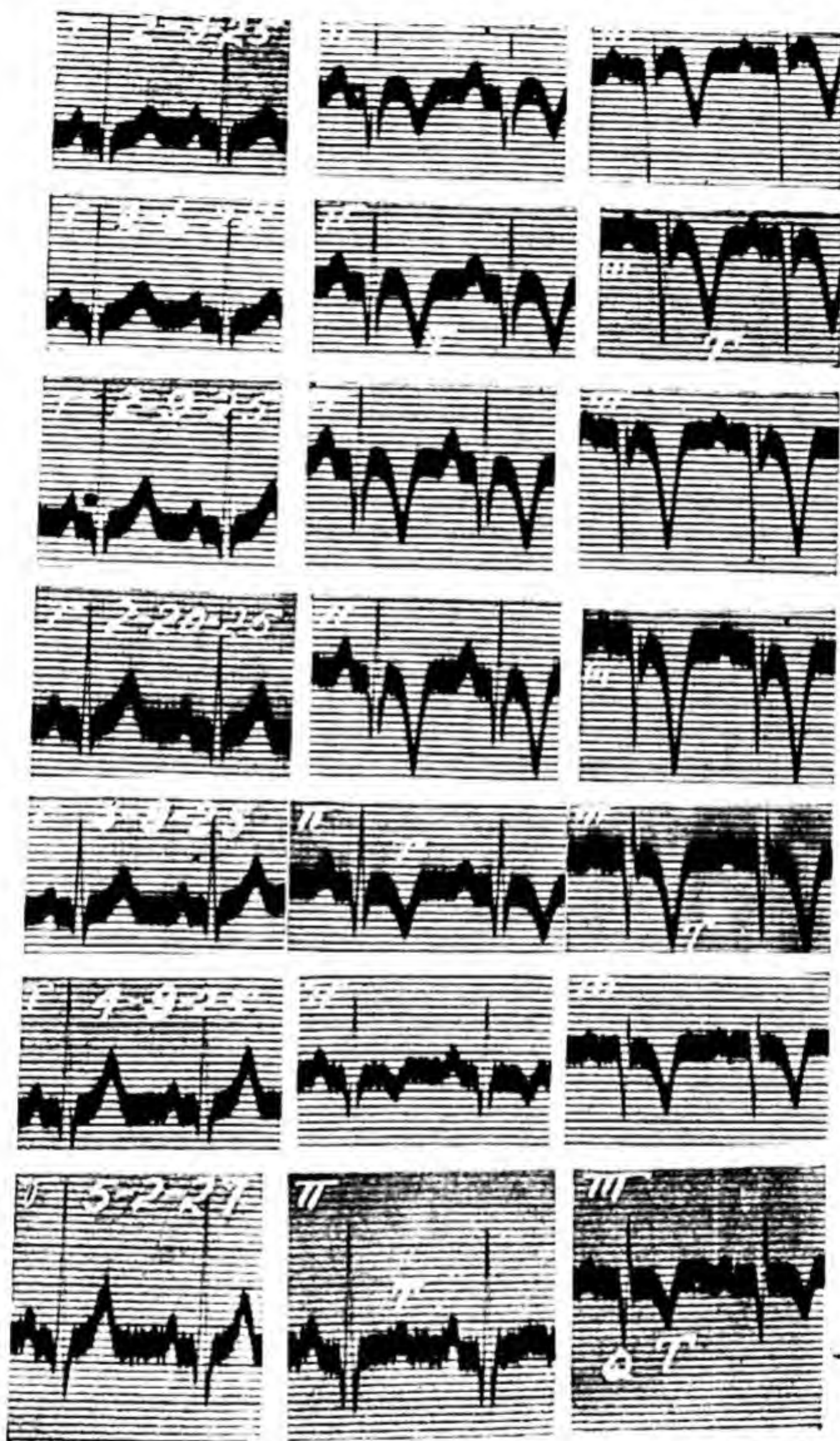


Fig. 111.—Coronary Type of Ventricular Complex (Posterior Lesion). Attack of coronary thrombosis on January 22, 1925. Note the sequence of changes in the ventricular complexes beginning with a high take-off of the R-T segment in Lead III, gradually becoming lower and dipped and finally marked inversion of T<sub>3</sub>. Partial heart block was present in the early days. A prominent Q<sub>3</sub> occurred and persisted. The patient did well for five years after the attack. These changes indicate infarction in the posterior aspect of the left ventricle. Similar progressive changes when present in Lead I indicate anterior infarction. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

or useful for the elucidation of certain occasional diagnostic problems, the routine procedure I am following at present is to obtain only one precordial lead. The single, most useful position seems to be from the apex of the heart to the left leg. It is also important to realize that



marked differences in the character of the complexes may result from slight changes in the position of the exploring electrode (Fig. 117).

In general there are three types of information to be obtained from precordial leads. During the early acute stage, the QRS-T segment may be conspicuously displaced upwards or downwards (Figs. 105, 115, 118). This generally accompanies significant alterations in the complexes of the three conventional leads. There are instances, however, in which the change in Lead IV may be the only evidence of an acute myocardial injury, the three leads showing nothing abnormal (Fig. 118). This sign, if it is to be obtained at all, will be more often found at the apex than

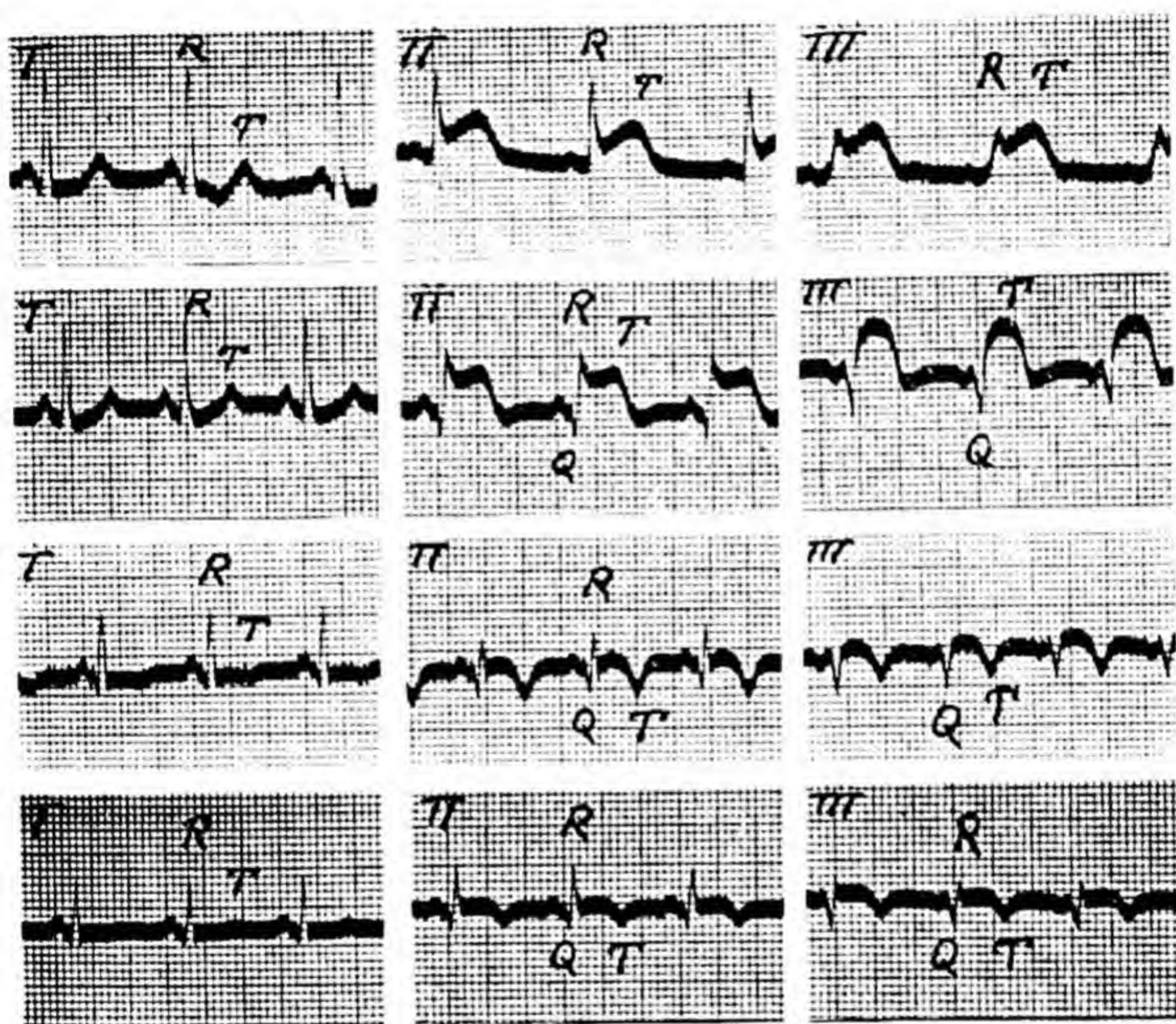


Fig. 112.—Coronary Type of Ventricular Complex (Posterior Lesion). Attack June 14, 1934, four hours before first set of curves. Subsequent tracings taken June 20, 1934, July 6, 1934, and August 17, 1934. Note high take-off of R-T interval in Leads II and III and progressive changes. A  $Q_s$  and  $Q_s$  gradually develop and persist.

elsewhere, but there may be instances in which other points over the chest will prove valuable. In order to detect changes in the posterior part of the ventricle, in some cases it would be necessary to insert an electrode into the esophagus. It would then lie in close approximation to the posterior portion of the ventricles. This is obviously a difficult procedure for patients who are critically sick, and will necessarily have limited application.

When the deviation of the R-T segment is upward it points to an anterior lesion (Fig. 118) and when it is downward to a posterior lesion (Fig. 115). Anterior or lateral infarction results, for the most part, from



a narrowing or occlusion of the left anterior descending coronary artery, and involvement of the posterior or diaphragmatic portion from a lesion of the left circumflex or the right coronary artery. There may be no displacement of R-T in Lead IV when it is quite prominent in one of the conventional leads (Fig. 107, third set of curves). These changes in Lead IV may occur within a few hours or days after the onset of the at-

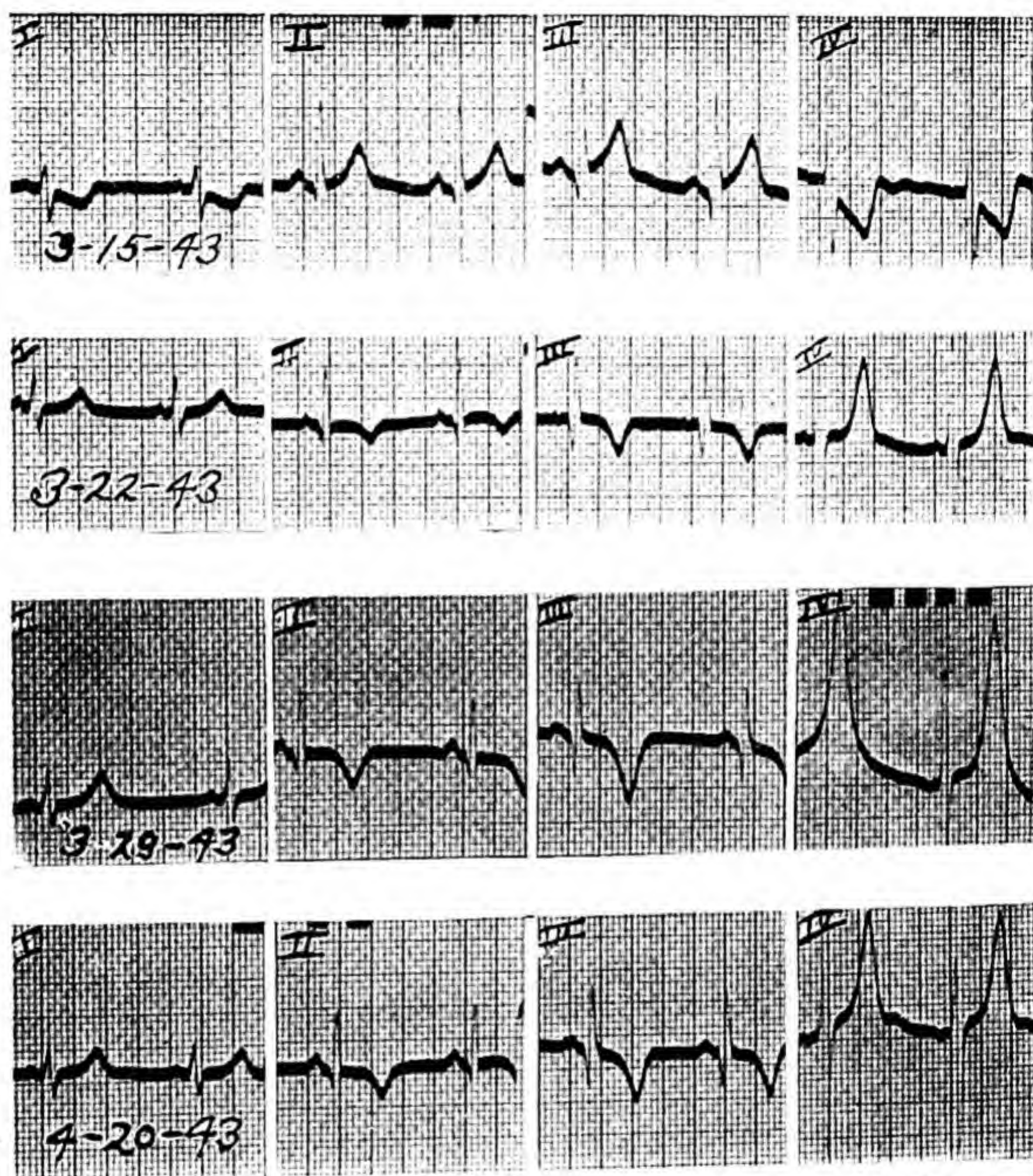


Fig. 119.—Abnormal form of Ventricular Complex (Posterior Infarction). The first set of tracings was taken six hours after the onset of a typical attack of coronary thrombosis. Note depressed S-T interval in Leads I and IV with elevation of S-T interval in Leads II and III, a distinct  $Q_1$  and  $Q_2$  and then the gradual development of sharp inversion of  $T_1$  and  $T_2$  with a marked exaggeration of the T wave in Lead IV. The patient was a man forty-two years old, who, while shoveling sand, had severe pain in the chest radiating down both arms. He had slight fever, leukocytosis, increased sedimentation rate and the blood cholesterol was 410. Recovery was excellent.

tack, but almost always disappear with healing and recovery. When the initial ventricular complexes are large, slight or even marked deviations of the R-T interval must not be regarded as absolutely diagnostic, as they occur without acute myocardial infarction (Fig. 119). There are instances of permanent deviation of the R-T segment that cannot be interpreted as evidence of an acute lesion (Fig. 120), for it may be present for



years. This is particularly true in cases of bundle branch block. Notwithstanding the above limitation, displacement of the R-T segment in Lead IV may be very valuable diagnostically.

The second aspect of Lead IV which may be helpful in diagnosis is the appearance of the T wave and whether it is positive or negative, upright or inverted. Normally,  $T_4$  obtained from an apex lead is upright, but an inverted  $T_4$  is not pathognomonic of myocardial infarction. It

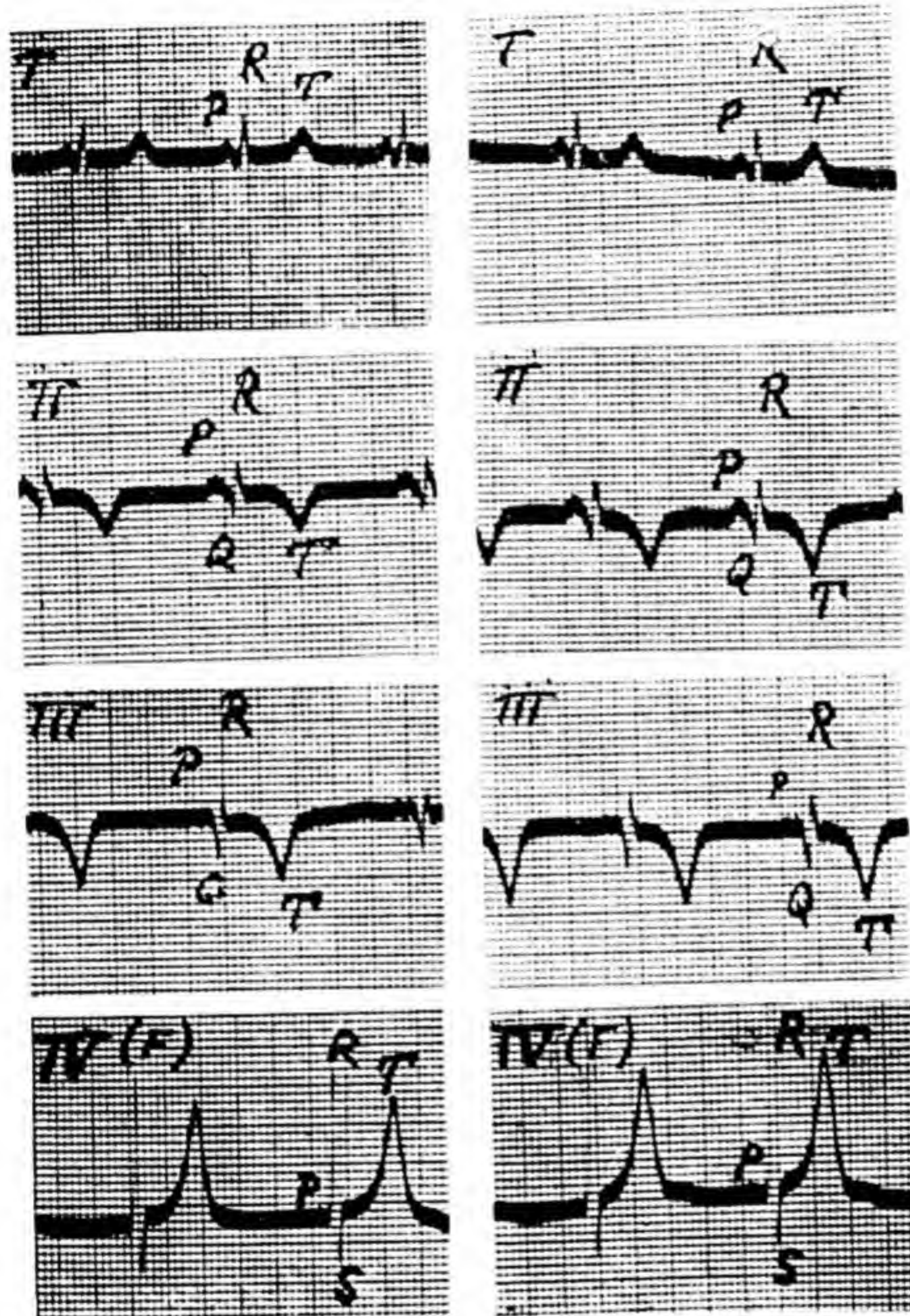


Fig. 114.—Coronary Type of Ventricular Complex (Posterior Lesion). The attack occurred June 24, 1934. The first tracing was made July 11, 1934, the second July 18, 1934. Note rounded and dipped  $T_1$  and  $T_2$ . Lead IV shows normal  $R_4$  but exaggerated upright  $T_4$ .

occurs in infants, in some cases of organic heart disease, such as mitral stenosis or hypertensive heart disease, and occasionally in other conditions. However, it is very common in infarction of the anterior portion of the left ventricle.

The acute changes in the R-T segment described above rapidly undergo alterations. The last portion of the complex is apt to be pointed and dipped (Fig. 118). During subsequent days there may be progressive inversion or increase in height of  $T_4$ , depending on whether the



original change was a high or a low take-off. Finally,  $T_4$  may become permanently inverted, return to normal or assume an intermediary abnormal form. In posterior lesions  $T_4$  does not become inverted and the only thing about it that might suggest the presence of such a lesion is a considerable exaggeration in height which is sometimes seen (Fig. 114). Therefore, in most cases of posterior lesions and in many anterior ones the final form of the T wave is normal or not characteristically abnormal.

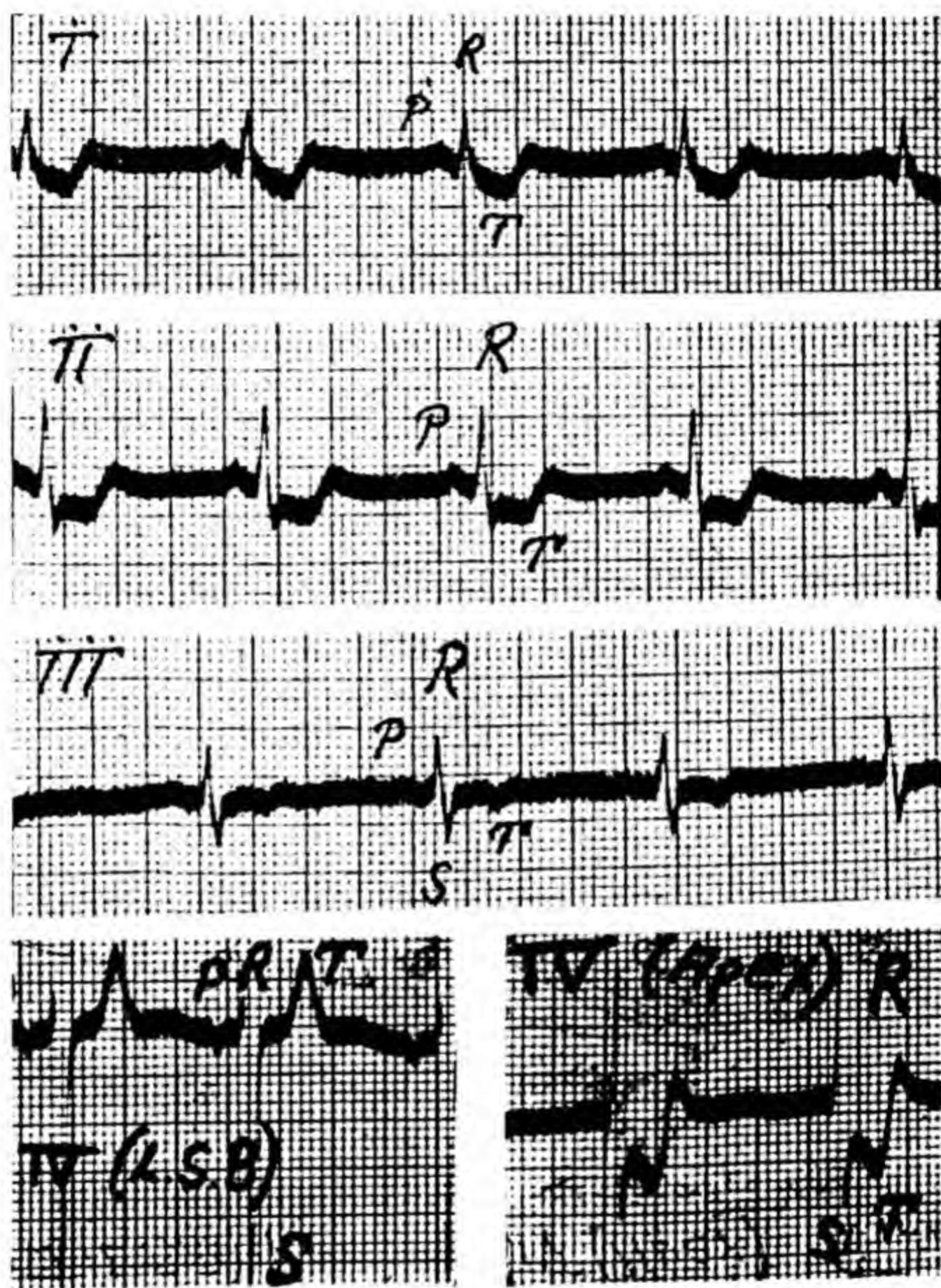


Fig. 115.—Coronary Type of Ventricular Complex (Posterior Lesion). Tracings taken a few minutes after the onset of an attack. Note low take-off of  $T_4$  in the apex lead. A high take-off occurs in the acute stages of an anterior lesion (compare Fig. 105).

The third change in Lead IV that pertains to the diagnosis of myocardial infarction is the form of the initial ventricular complex (formerly called  $Q_4$  but now called  $R_4$ ). Practically all normal hearts will show an initial upward deflection ( $R_4$ ) of some magnitude, when curves are obtained from an apex lead. Occasionally a small downward deflection of a few millimeters or less may precede the normal tall  $R_4$ . An acute injury of the anterior part of the left ventricle, such as an infarction, often leads to a complete disappearance of  $R_4$ . In fact it is extremely rare to find such a change in any other condition or even in other forms of heart dis-



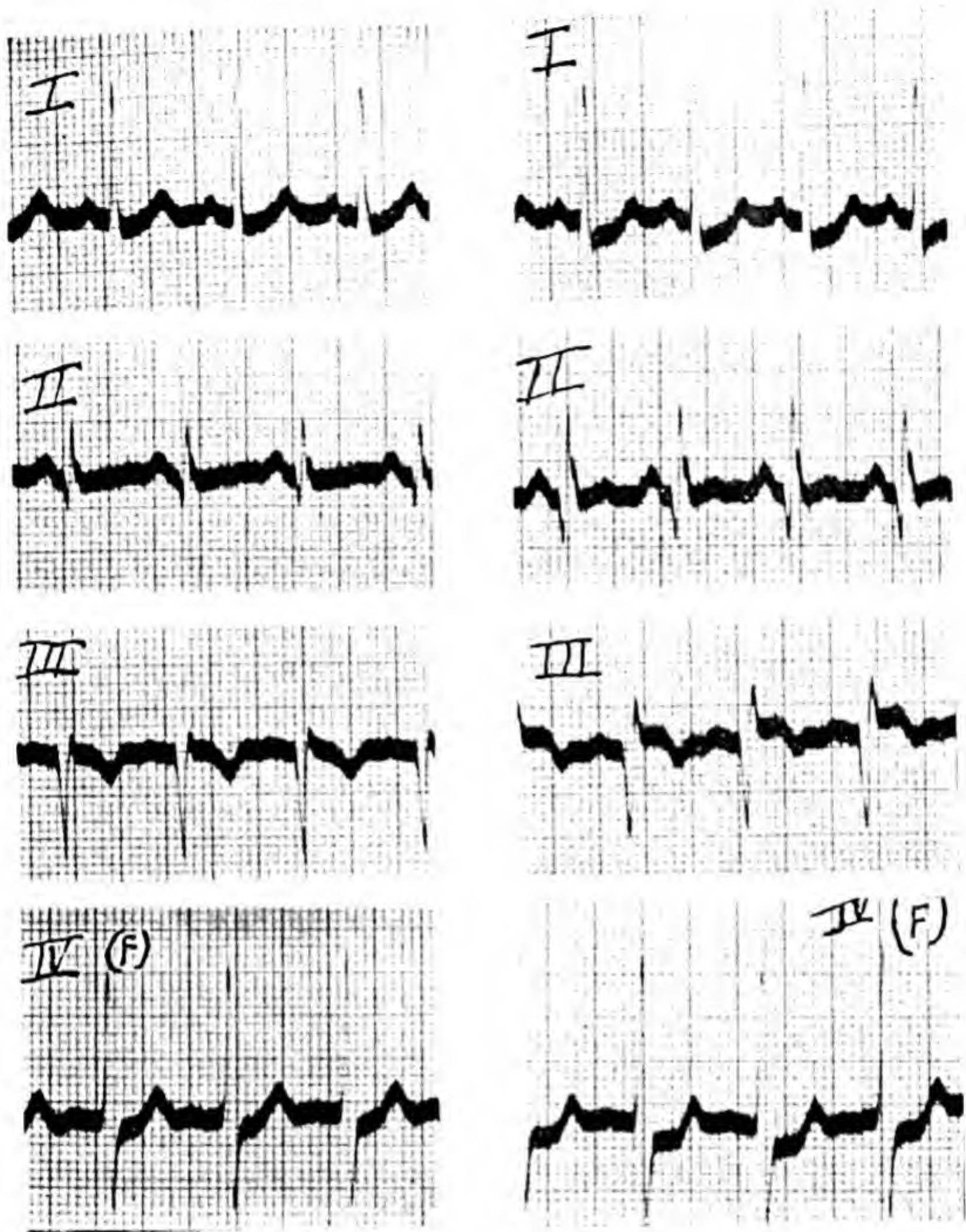


Fig. 116.—Abnormal Form of Ventricular Complex (Produced by Exercise). The patient was a young man thirty-five years old who complained of pains in both arms, only on effort. The physical examination revealed no abnormality. The first set of curves was somewhat abnormal but not conclusive. The second set, taken five minutes later after briskly walking up four flights of steps, shows conspicuous depression of S-T segment in Lead I and Lead IV and elevation of S-T. This identified the symptoms as coronary in origin.

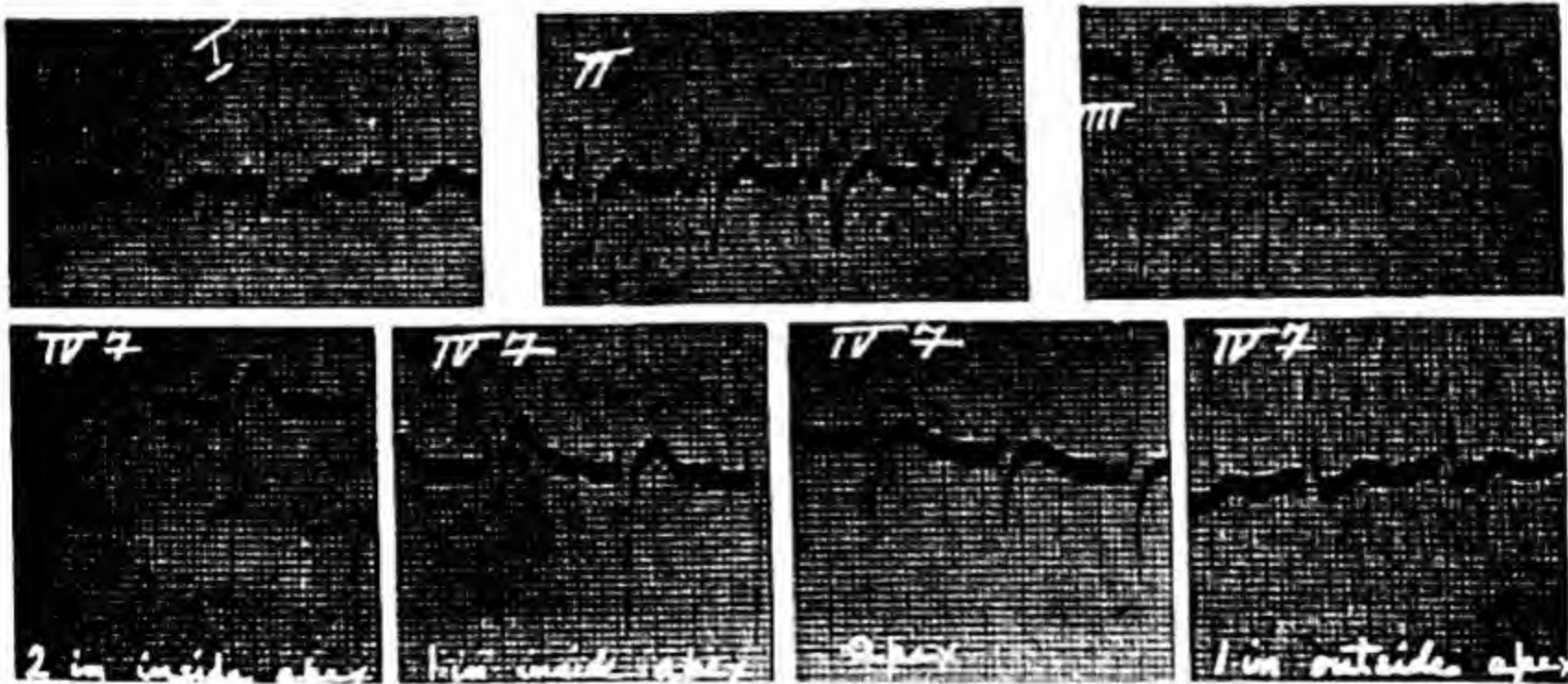


Fig. 117.—Effect of Change of Position of Exploring Electrode on Lead IV. Note marked variations in ventricular complex as electrode is moved from inside to outside apex impulse.



ease. It occurs usually in the early days following an acute coronary thrombosis, but not in posterior lesions. Once  $R_4$  disappears it tends to be a permanent change. Although occasionally it returns, it is then apt to be of comparatively low amplitude. The changes in the initial ventricular complexes of Lead IV and of the other three leads are much more likely to persist indefinitely than those in the T wave. In this way the absence of  $R_4$  may be very valuable in the diagnosis of an anterior lesion that occurred weeks, months, or even many years before (Figs. 106 to 110). I have seen an instance in which this sign persisted twenty-five years after an acute coronary thrombosis. The patient had remained

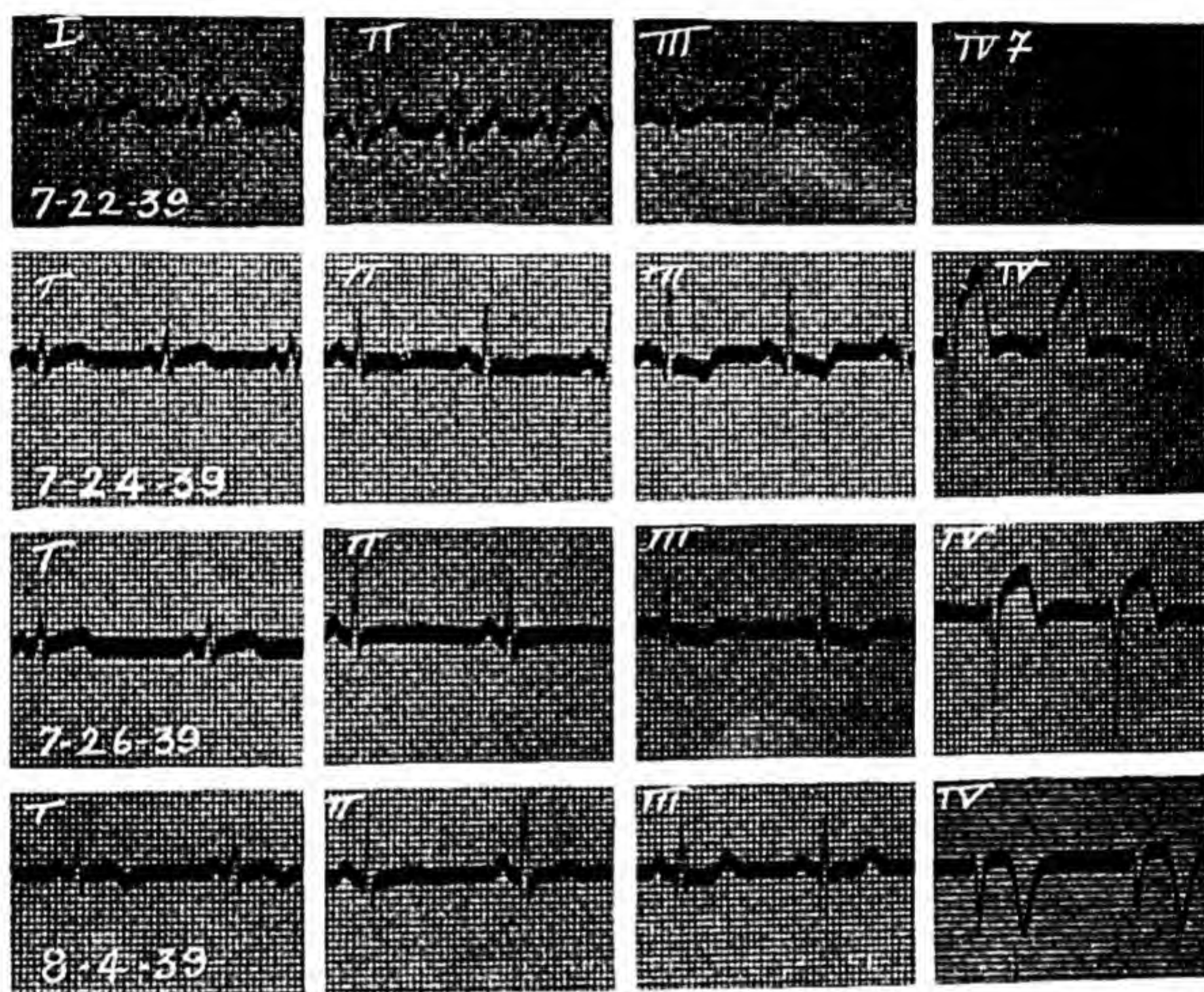


Fig. 118.—Acute Coronary Changes in Lead IV Only. Note that in the first set of curves the ordinary three leads are normal while Lead IV shows a high S-T segment. In subsequent curves significant changes in various leads develop.

in good health all those years, experiencing only an occasional attack of angina.

When left bundle branch block is present, there is no satisfactory electrocardiographic method of recognizing acute myocardial infarction. Occasionally, in serial curves, suggestive alterations in the form of the T wave and R-T segment may be present in Lead IV. With right bundle branch block, however, it is possible to obtain electrocardiographic evidence of an anterior but not of a posterior infarction. The appearance of a large  $Q_4$  or a W-shaped QRS complex in a precordial lead from a point between the nipple and the midsternal line would point to an anterior lesion. Inversion of  $T_4$  also takes place. Such changes almost never occur with right bundle branch block without myocardial infarction.



There are numerous instances in which  $R_4$  does not disappear entirely, but is small, measuring 2 mm. or less. This may be looked upon as suspicious but not unequivocal evidence of myocardial infarction, when found in a lead taken from the apex. It would not have very much significance in leads taken near the sternum because normally  $R_4$  is often quite small in tracings from these areas. In a considerable group of patients examined postmortem in whom electrocardiograms had shown a small  $R_4$  (apex lead), about one-half had coronary artery disease with old

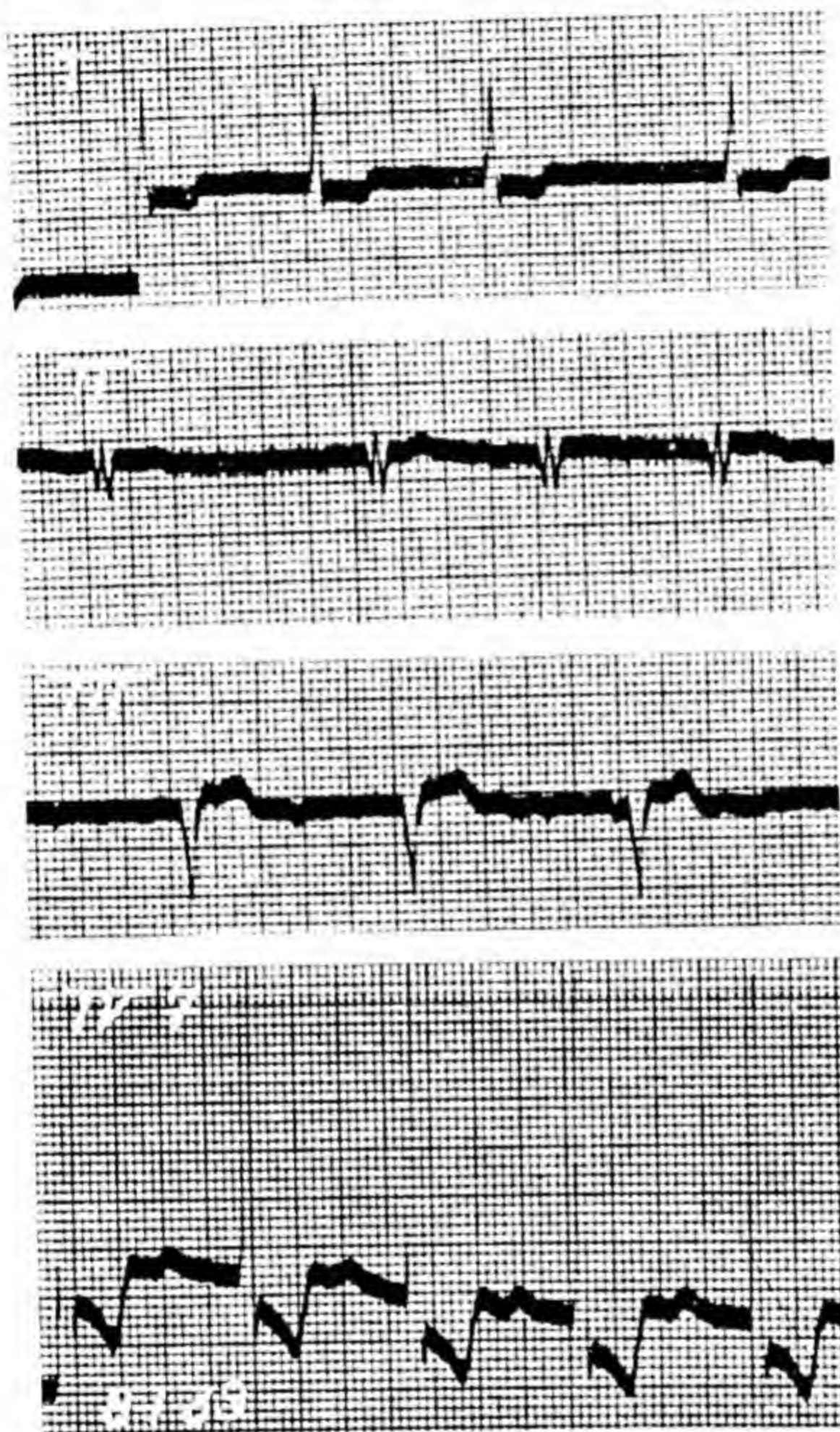


Fig. 119.—Depressed R-T<sub>4</sub>. Note marked depression of R-T<sub>4</sub>. Postmortem examination showed no acute myocardial infarction. There were small, multiple, old scars. The left descending coronary artery was markedly narrowed by an old arteriosclerotic process.

or recent myocardial lesions. The remainder had various other forms of heart disease and a few had no significant disease of the heart. When such small R waves in Lead IV are associated with an inverted T<sub>4</sub>, however, it is presumptive evidence of coronary artery disease.

In summary, it can be stated that precordial leads are a necessary aid in the diagnosis of myocardial disease. During the acute stage of coronary thrombosis they may furnish convincing diagnostic evidence whether the lesion is anterior or posterior. They frequently afford fairly decisive



proof of the existence of a previous anterior infarction, but only rarely help in the diagnosis of old posterior lesions.

The changes in the form of the ventricular complex in the four leads described in the preceding paragraphs are of great importance in the care of patients with disease of the coronary arteries. They often are distinctive and diagnostic. At times they are the sole data on which a

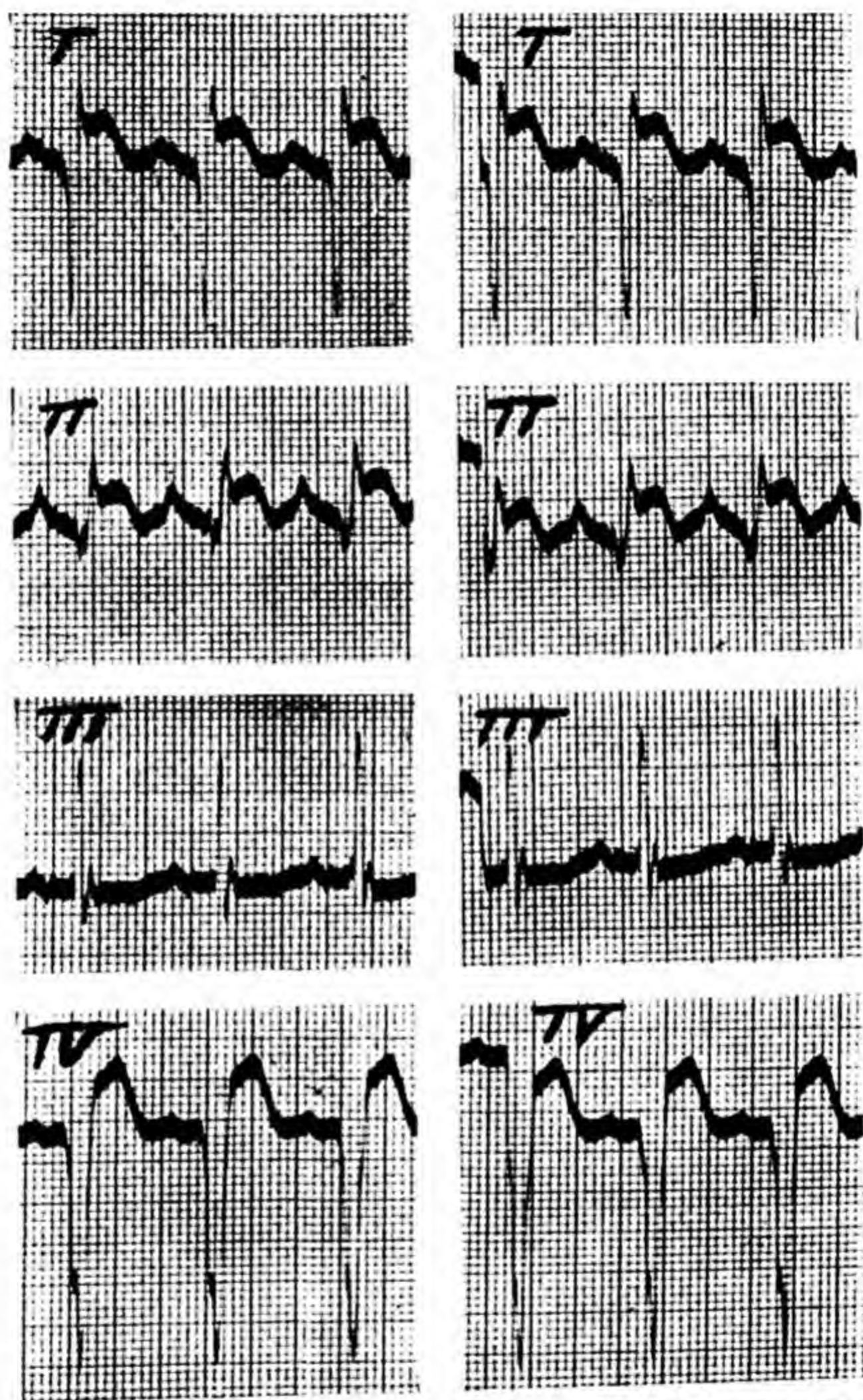


Fig. 120.—Elevated R-T<sub>4</sub>. The first four leads were taken May 15, 1938; the second set about one year later. The patient has hypertensive heart failure, but has had no clinical episodes of acute coronary thrombosis. Note the persistent marked high take-off of RST interval in Leads I, II, and IV.

diagnosis can be made. One set of curves taken at a particular time may be entirely normal or equivocal, but a series of several tracings during the first ten days following an attack of coronary thrombosis will almost invariably show sufficient evidence to establish a correct diagnosis. There are few other conditions besides myocardial infarction that are associated with such striking changes in the form of the ventricular complex from day to day. One may say that not only are certain specific al-



terations of the electrocardiogram diagnostic, but that marked variations over a short period of time are also helpful. One needs to become familiar with the various peculiar complexes because the first and possibly the only curve available may be one taken several days or weeks after the

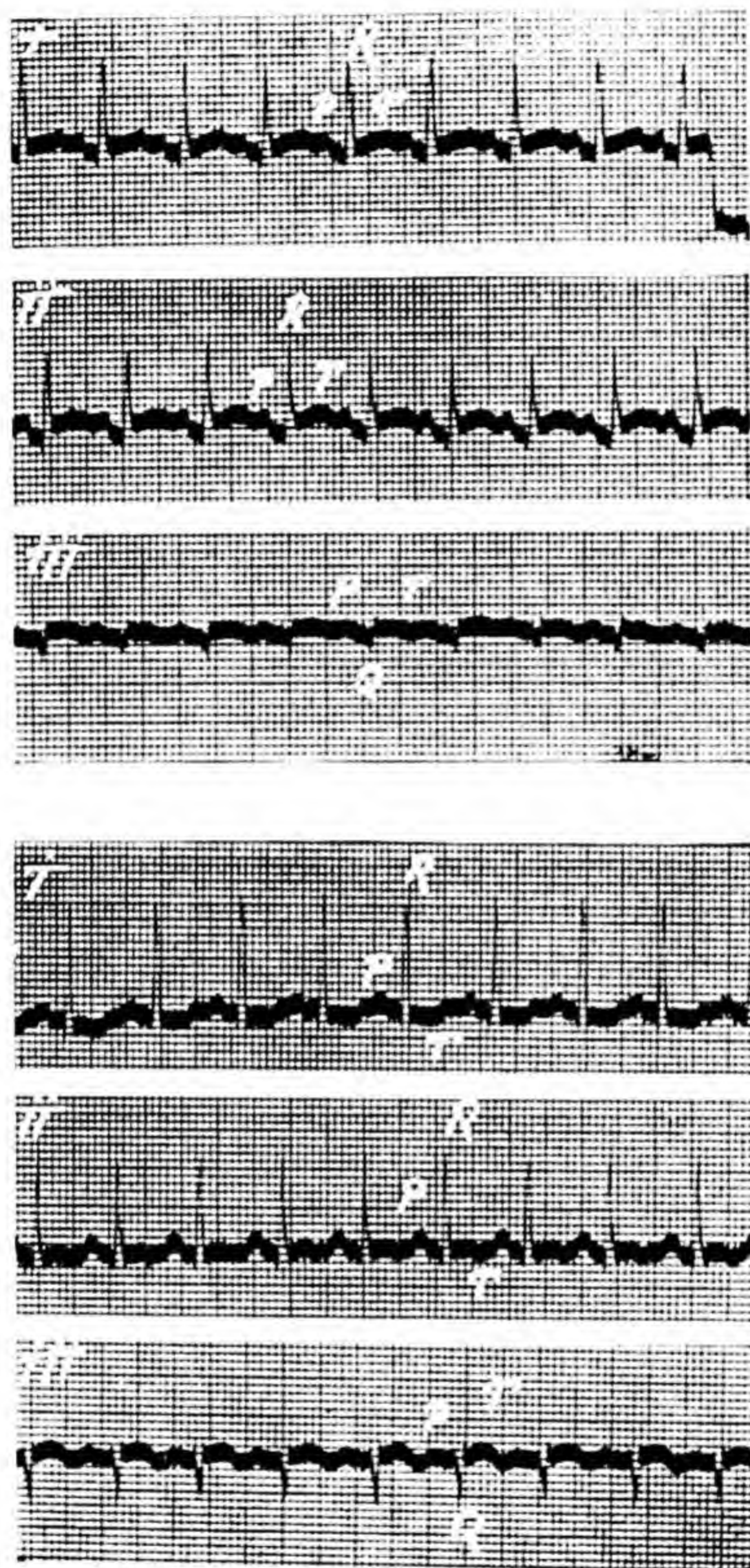


Fig. 121.—Abnormal Form of Ventricular Complex (Rheumatic Type). Note the slight elevation of the R-T segment in the upper curves taken May 16, 1935. These changes had practically disappeared on June 20, 1935, when the second set was taken. The patient had acute rheumatic pericarditis with effusion. The R-T changes resemble the coronary type to some extent.

attack. Suppose that tracing happened to be the fifth shown in Figure 111 (taken on January 28, 1925). It must be recalled that the period of the high take-off of the R-T segment has passed and that the marked inversion has not yet taken place. One should try to visualize the various



changes in relation to the date of the attack. Furthermore, in many cases only slight but, nevertheless, significant deformities of the complexes are shown. When in doubt it will be necessary to obtain repeated electrocardiograms to decide whether these changes are of importance.

Because of the increasing role that precordial electrocardiography is playing in the diagnosis of coronary artery disease and other conditions,

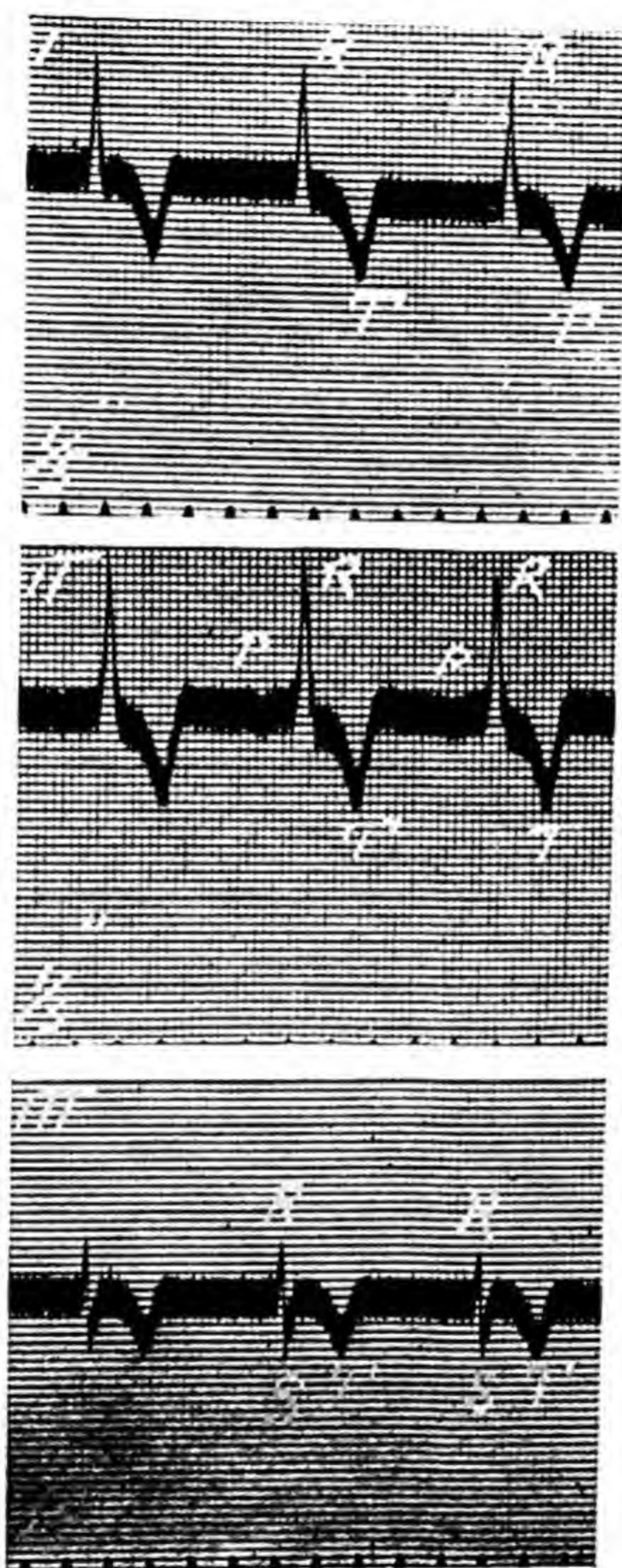


Fig. 122.—Abnormal Form of Ventricular Complex. Note that the QRS complex is not broadened but that the T waves are markedly inverted in all leads. This patient died of fibrous myocarditis several weeks later. (Author's article in Oxford Loose-Leaf Medicine, vol. II Courtesy of Oxford University Press.)

a more detailed discussion of its theoretical and practical aspects will be taken up at the end of this chapter.

The great diagnostic value of electrocardiography is well illustrated by several of these examples. Figure 105 shows an instance in which several competent observers had made the diagnosis of "fever, unknown origin." The man complained of weakness and sweats; there was no pain



in the chest or dyspnea. On very close questioning it was learned that he had a slight discomfort in the sternum. An electrocardiogram was taken because I had noted that many patients with coronary thrombosis had sweats. This established a definite diagnosis of acute coronary thrombosis without pain. Another patient (Fig. 108) had an attack three weeks before he was seen, which was called "gastritis." The electrocardiogram proved that the attack was a recent coronary thrombosis. The case illustrated by Figure 109 was puzzling because it was known that the patient had an old peptic ulcer and had been under treatment for it for many years. On this day he came to the gastro-intestinal clinic, as was his custom, but complained of a somewhat different squeezing distress in the lower sternum which began several months before. The ordinary three leads could only be regarded as suggestive, but the fourth lead was pathognomonic of an anterior infarction. Finally, it has been found that patients with angina pectoris, who never had an acute major episode that required them to go to bed or that could be recognized clinically as

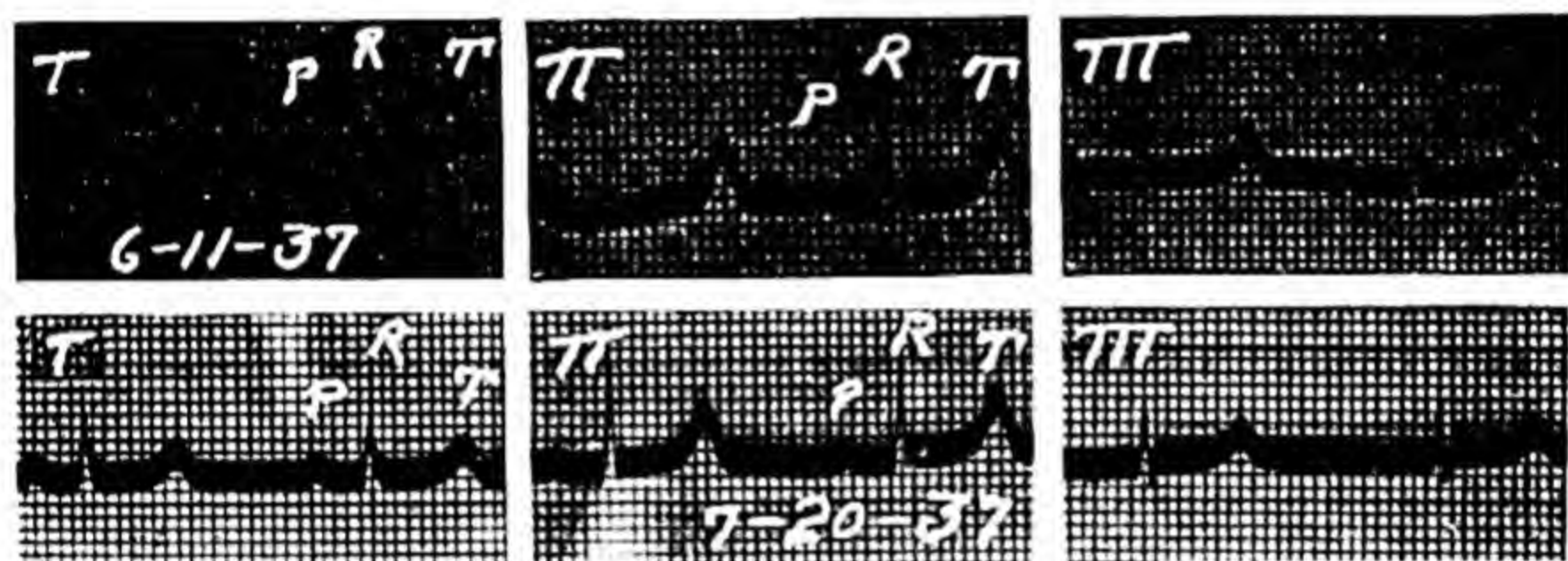


Fig. 123.—Prolonged R-T Interval in Hypoparathyroidism. The patient was a man sixty-eight years old with hypoparathyroidism. Blood calcium on June 11, 1937, was 4.7 milligrams and on July 20, 1937, it was 9.3 milligrams. The R-T interval decreased from 0.48 second to 0.40 second. The improvement occurred as a result of dihydrotachysterol.

coronary thrombosis, not infrequently have curves that are diagnostic of myocardial infarction (Fig. 110). From this it follows that myocardial infarcts may occur as a result of gradual narrowing of the coronary vessels without acute thrombosis or that some of the attacks of angina are, in fact, attacks of thrombosis. Both of these possibilities are supported by postmortem experience.

**Ventricular Complexes in Rheumatic Fever.**—The frequency of conduction defects in rheumatic fever was mentioned previously. There are also minor changes in the form of the ventricular complex that slightly resemble those seen in myocardial infarction. They are quite common, especially if the rheumatic infection of the heart is associated with acute pericarditis with or without pericardial effusion. These changes consist of a slightly high take-off of the R-T segment or a loss of the iso-electric interval between the R and T waves (Fig. 121). The displacement of the R-T segment is concordant (all in the same direction) in rheumatic fever, whereas in coronary thrombosis it is discordant (upwards in one lead



and downwards in another). One does not see such marked changes as in coronary thrombosis nor the striking inversion of the T wave and as yet it has not been found that  $R_4$  disappears, although Lead IV may show inverted T waves or deviations of the R-T segment. The changes noted in rheumatic carditis disappear during the course of weeks. Occasionally these findings will serve as important additional evidence for or against

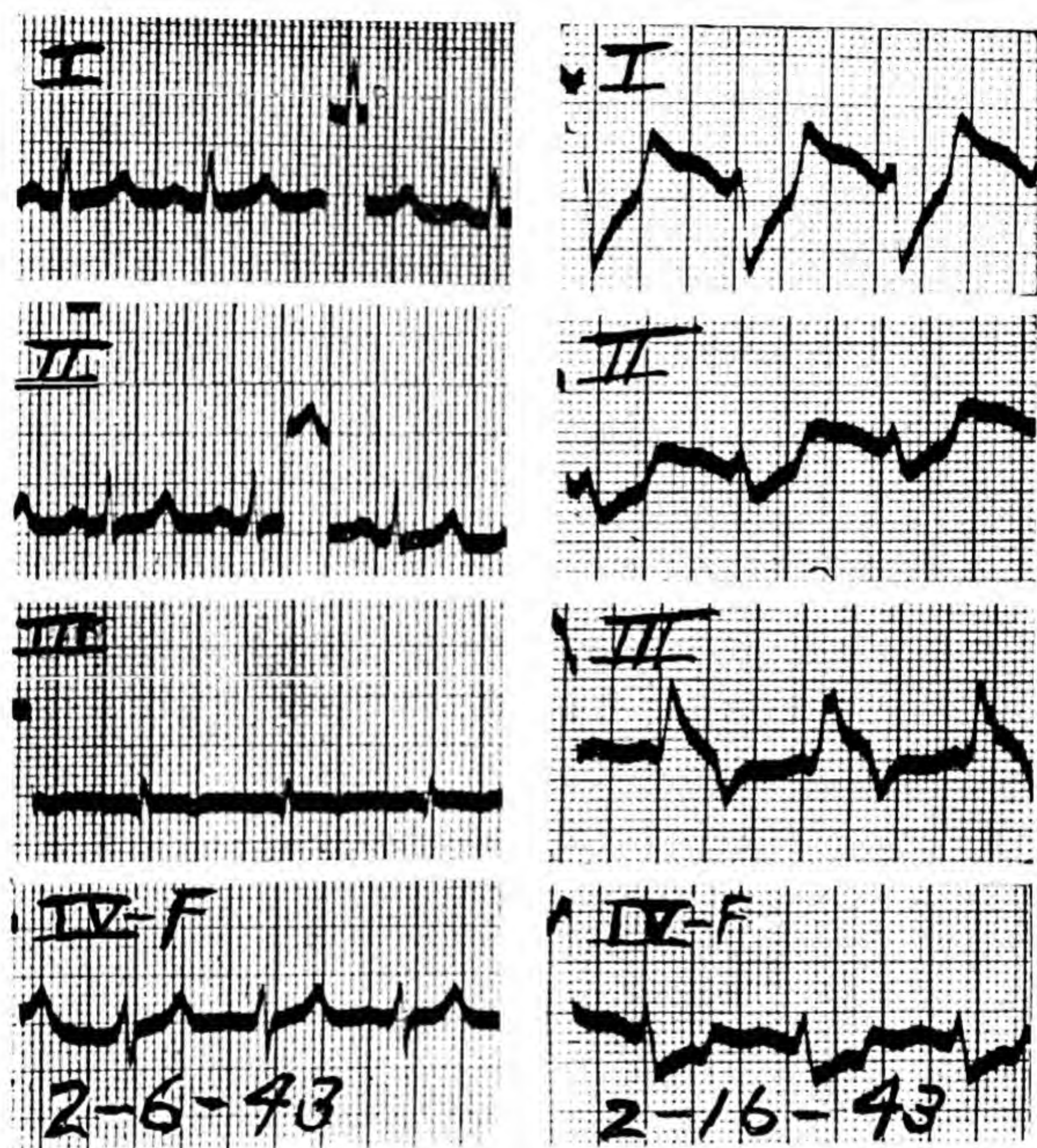


Fig. 124.—Abnormal Form of Ventricular Complex (Potassium Poisoning in Uremia) The first set taken February 6, 1943, shows essentially normal complexes of slightly low amplitude. The second set, taken ninety minutes before the patient's death on February 16, 1943, shows inconspicuous P waves and markedly deformed QRS and T waves. The serum potassium at this time was 9.2 millequivalents per liter (normal = 4 to 5 millequivalents per liter). The patient had advanced chronic nephritis and uremia.

the diagnosis of rheumatic fever as an explanation of an otherwise obscure illness.

**Ventricular Complexes in Other Conditions.**—There are numerous other conditions in which the ventricular complex alters its form. In many the changes are not sufficiently characteristic or uniform to be diagnostic. Further study will be necessary before such data can be standardized and before decisive conclusions can be drawn. In the meantime it



is important to be familiar with some of the peculiarities that may arise. Figure 122 illustrates a set of curves that do not conform to any simple classification and yet one can be sure that they represent a pathological heart muscle. Markedly inverted T waves in all leads indicate a poor prognosis. This patient died of fibrous myocarditis several weeks later. Some of these markedly abnormal ventricular complexes probably represent multiple areas of healed myocardial infarctions, some of which cases are described pathologically as fibrous myocarditis.

In uremic states bizarre and abnormal ventricular complexes will be found. The Q-T interval may be abnormally lengthened and at that time the calcium content of the blood may be considerably decreased. In rare cases of uremia the P waves gradually decrease in size and finally dis-

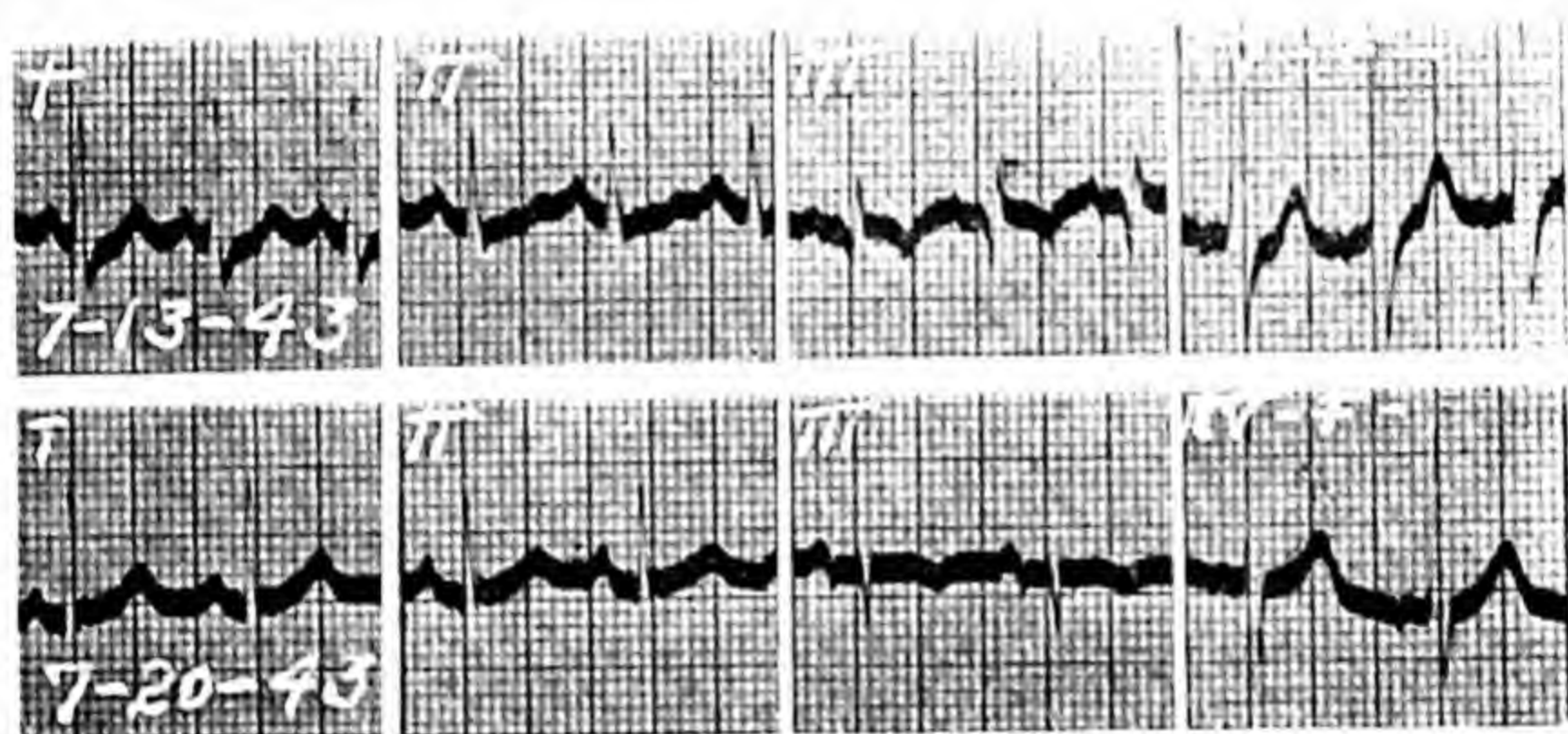


Fig. 125 — Abnormal Form of Ventricular Complex (Pulmonary Embolism) The patient was a man forty-two years old, who was well except for an inguinal hernia. His heart was normal. Operation was performed on July 1, 1943, under spinal anesthesia. He was doing well when on July 13, 1943, while on a bed pan, he had sudden pain in the left anterior chest, dyspnea, tachycardia and apprehension. The pulse rate was 140, respiration 28, blood pressure 180 systolic and 80 diastolic. His legs showed nothing abnormal. x-Ray showed slightly cloudy left base and elevated left diaphragm. On July 14, 1943, bilateral ligation of the femoral veins was performed. The patient ran a temperature of 101° to 102° F. for five days and recovered. The first tracings show changes indicative of acute cor pulmonale, *i.e.*, an  $S_1$ , a  $Q_3$ , a depressed  $S-T_1$ , an elevated  $S-T_3$ , and an inverted  $T_3$ . Note the disappearance of these changes in the second tracing.

appear and the QRST complex becomes very abnormal, with marked spreading of the QRS waves and distortion of the S-T segment (Fig. 124). These latter changes are regarded as due to potassium poisoning of the heart, as the content of potassium in the blood is strikingly elevated when such abnormalities are found.

Pulmonary embolism with or without pulmonary infarction not infrequently produces peculiar changes in the ventricular complexes. These consist of a slight depression of the S-T segment in Lead I and slight elevation in Lead III, a definite  $S_1$  and possibly a  $Q_3$ . The T wave may be slightly inverted in any of the three leads. The changes that occur disappear when recovery takes place (Figs. 125, 126, 127).

Flattening of the T wave and decrease in the height of the QRS complex also occur in constrictive pericarditis (Fig. 128), marked anemia,



emphysema, beri-beri heart (Fig. 129) and myxedema. The Q-T interval, which is thought by some to be lengthened with myocardial fatigue, can be distinctly increased with tetany. I have seen an instance of marked lengthening of the Q-T interval in a case of tetany following pyloric obstruction and constant vomiting in which the  $\text{CO}_2$  combining power of the blood was about 100 volumes per cent. Whether the change in the

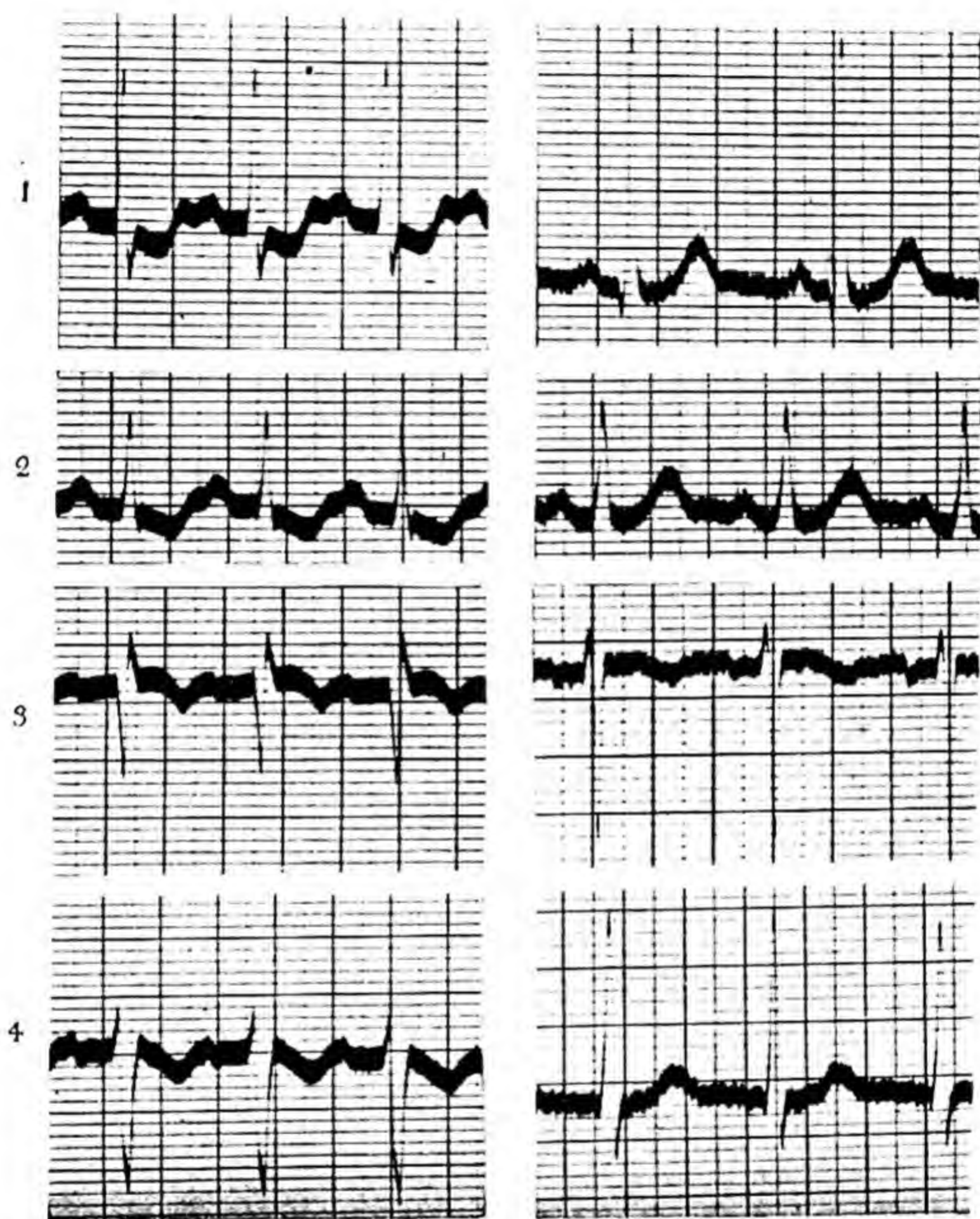


Fig. 126.—Abnormal Form of Ventricular Complex (Pulmonary Embolism). The patient was a woman nineteen years old, who had repeated pulmonary emboli post-partum several days before the first tracing was made. Note  $S_1$ ,  $Q_3$ , depressed and inverted  $S-T_1$ , and slightly elevated  $S-T_3$ . These changes are not present in tracings made March 3, 1943. (Courtesy of Dr. Paul D. White.)

Q-T interval was due to the abnormal acid-base equilibrium of the blood or to a disturbance in calcium metabolism was not clear. The Q-T interval is also increased in hypoparathyroidism (Fig. 123). The average normal Q-T interval can be determined from the following formula:  $K = \frac{Q-T}{\sqrt{R-R}}$ .

The normal figures for K are 0.39 for men and 0.44 for women.

Changes in the electrocardiograms are not rare during diphtheria. Younger patients are more apt to show prolongation of the P-R interval and more advanced stages of heart block, while the older more fre-



quently show alterations in the R-T complex such as a depression in the S-T segment or inversion of the T wave.

Inversion of the T wave in Lead I or Lead II is not normal and yet its presence must not always be regarded too seriously. When the inversion

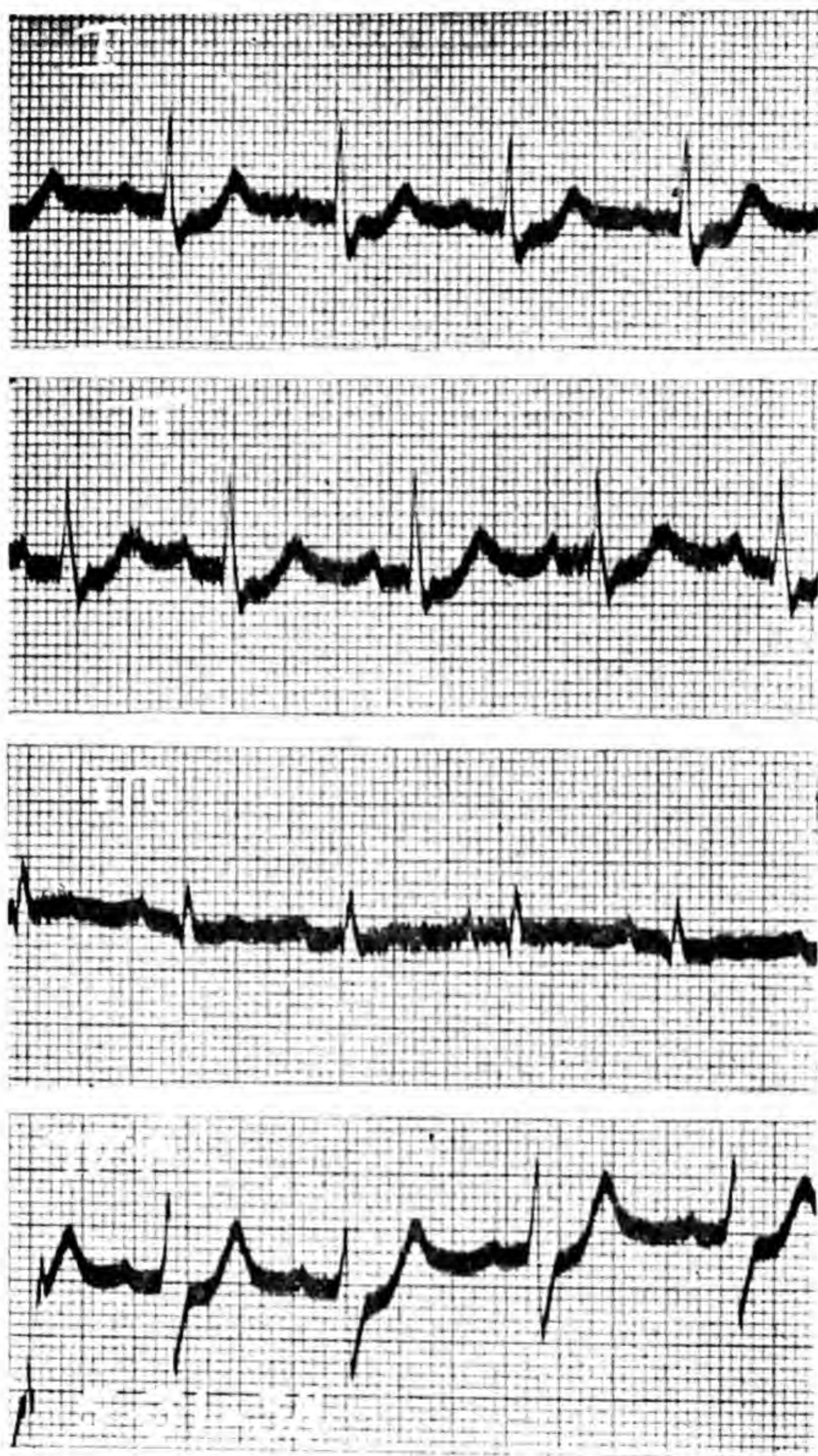


Fig. 127.—Abnormal Form of Ventricular Complex (Pulmonary Embolus). A woman sixty-three years old had a resection of the stomach for cancer and was convalescing well. Eighteen days after the operation while walking around the ward she collapsed suddenly but experienced no pain. She became cyanotic, dyspneic, lost consciousness and died in two hours. Her blood pressure was 50 systolic and 20 diastolic. Tracing was made one hour after the onset. Note the slightly depressed S-T segment in Leads I, II and IV with well-marked  $S_1$  and  $S_2$  and slight  $Q_1$ .

has the peculiar form described under myocardial infarction it has important significance. But the type shown in the lower set of curves of Figure 130 is frequently found in patients with hypertension but without any



important cardiac disease. In general as the R wave becomes taller and greater in area the T wave becomes smaller and inverted. Although this may indicate a change in the ventricular musculature, it cannot be regarded in a clinical sense as signifying myocardial disease. It must be recalled that the T waves can be diphasic or slightly inverted in neuro-circulatory asthenia. Cooling the apex of the heart by drinking cold water can also produce inversion of the T wave. Nitroglycerin or amyl nitrite can temporarily correct an inverted T wave in disease of the coro-

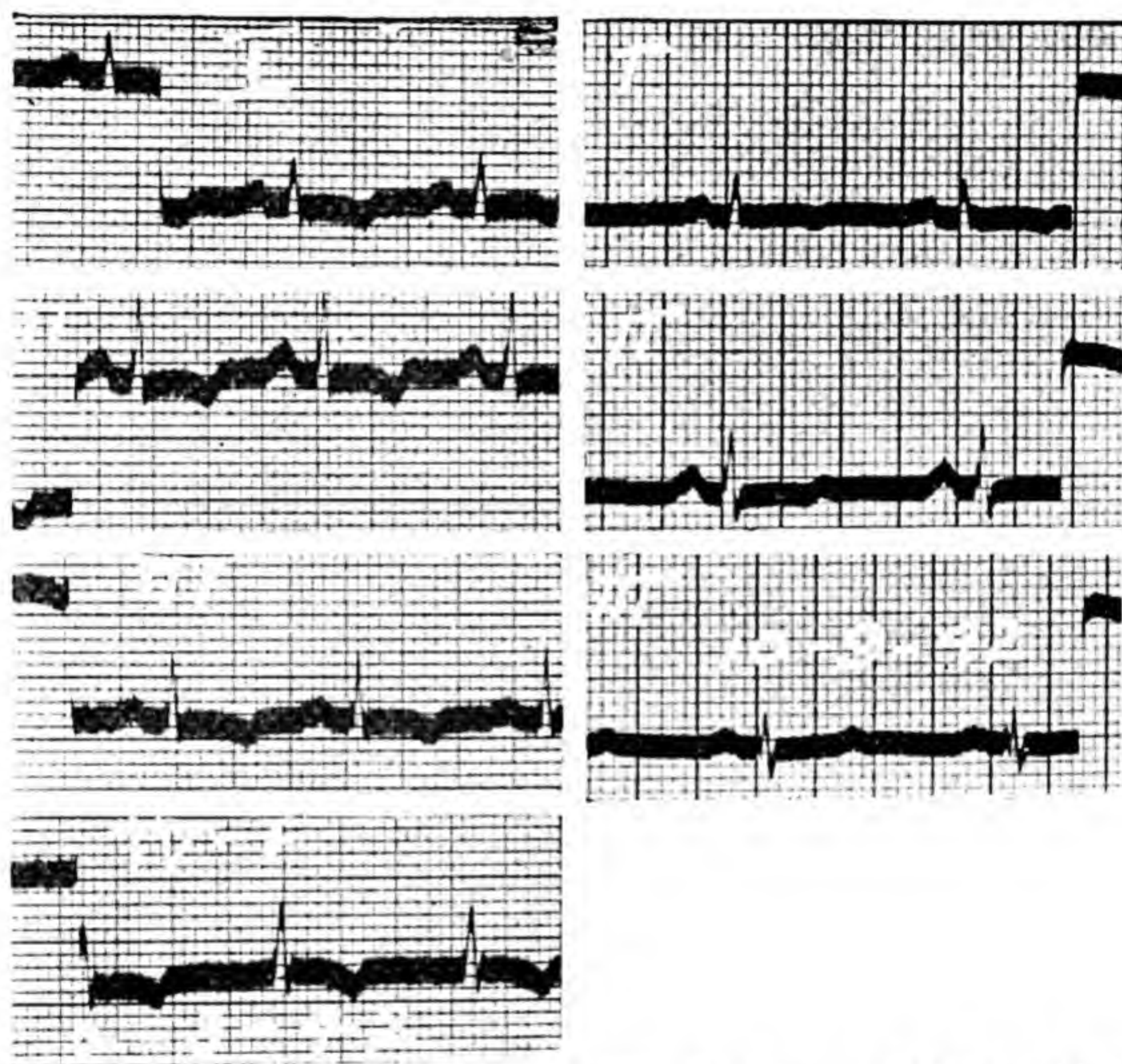


Fig. 128.—Abnormal Form of Ventricular Complex (Constrictive Pericarditis). The patient was a woman thirty-three years old who complained of fatigue, dyspnea and swelling of the face. Examination showed a quiet heart, diminution of amplitude of ventricular contractions, calcification of the pericardium, edema of the face, and increased venous pressure. Resection of the pericardium was performed by Dr. E. C. Cutler on February, 1942. Recovery was satisfactory and several months later she was working full time. The first set of tracings taken February 3, 1942, show rather low QRS waves and inverted T waves in all leads. The second set taken October 9, 1942, show slight restoration of the T waves to a more normal form.

nary arteries, and attacks of angina pectoris or a simple muscular effort may bring out inversion of the T wave in certain individuals. It is obvious that abnormalities of the ventricular complex are frequent and require cautious interpretation.

There is one peculiar type of electrocardiogram that occurs in a small group of individuals who are prone to have attacks of paroxysmal tachycardia (Fig. 131). This consists of a shortened P-R interval measuring less than 0.1 second and a broadened QRS complex to about 0.1 second.



Curiously enough, during the attacks of rapid heart action the QRS complex may be of normal duration. The explanation of this mechanism is

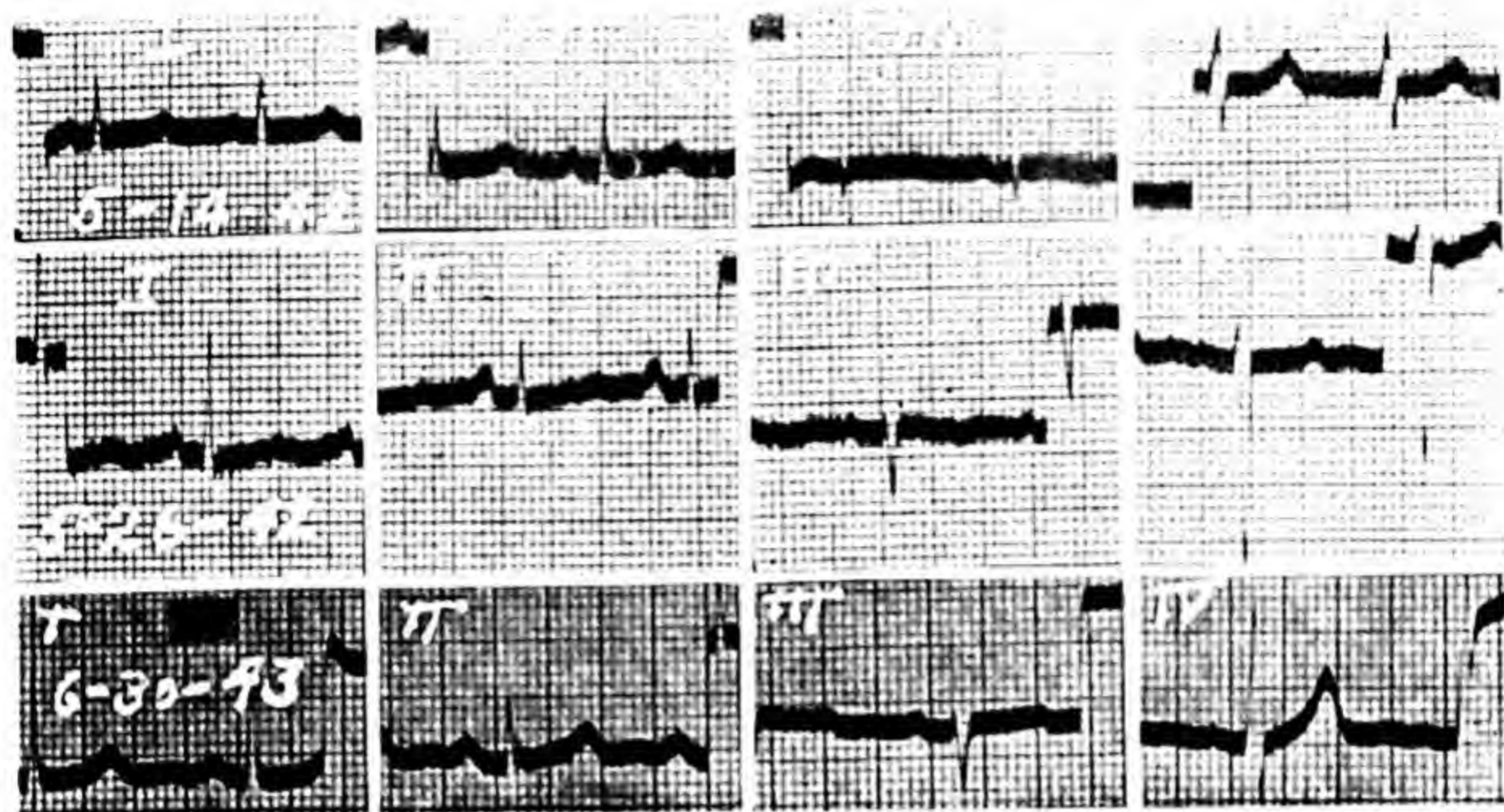


Fig. 129.—Abnormal Form of Ventricular Complex (Low Voltage in Beri-beri Heart) The patient was a man thirty-three years old, who had been drinking heavily and eating very little. He complained of weakness, shortness of breath, swelling of the legs and pains in the limbs. He showed marked dilatation of the heart, pulmonary congestion and some peripheral edema. A control period of digitalis caused no improvement. On a course of thiamine therapy the heart returned to normal size and all congestion disappeared. The first set of tracings (before thiamine) shows very low QRS complexes, which even in twelve days return to normal.

obscure. From a clinical point of view such patients may carry on normally and apparently have no organic heart disease. Although the early cases of this type were described as having paroxysms of auricular tachy-

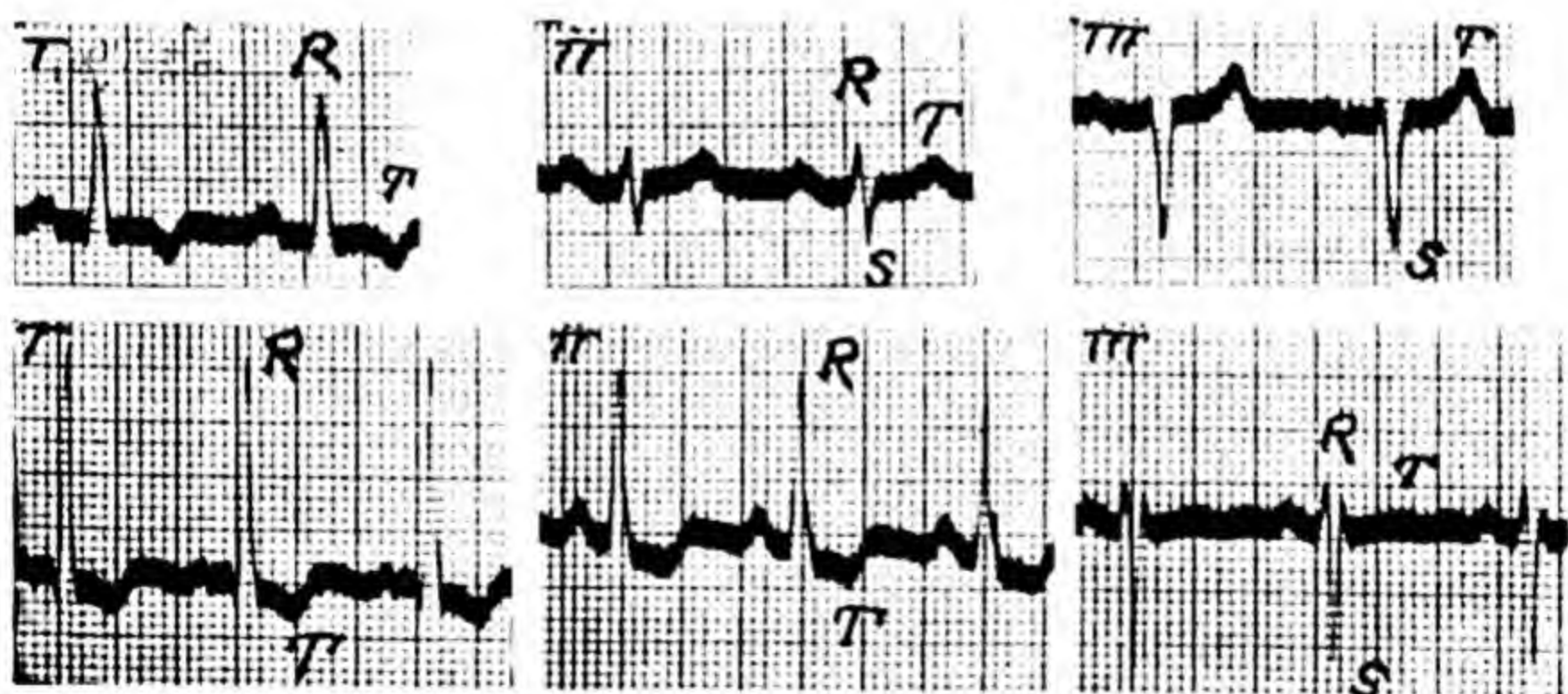


Fig. 130.—Abnormal Form of Ventricular Complex. Two sets of curves showing T waves that are inverted or diphasic in Leads I or II. The upper patient had hypertension and angina; the lower had hypertension without any cardiac disability. The clinical significance of such changes is often puzzling.

cardia, I have recently seen instances in which the attacks of rapid heart action were auricular fibrillation and others in which they were ventricular tachycardia (Fig. 132). Whenever curves showing these short P-R



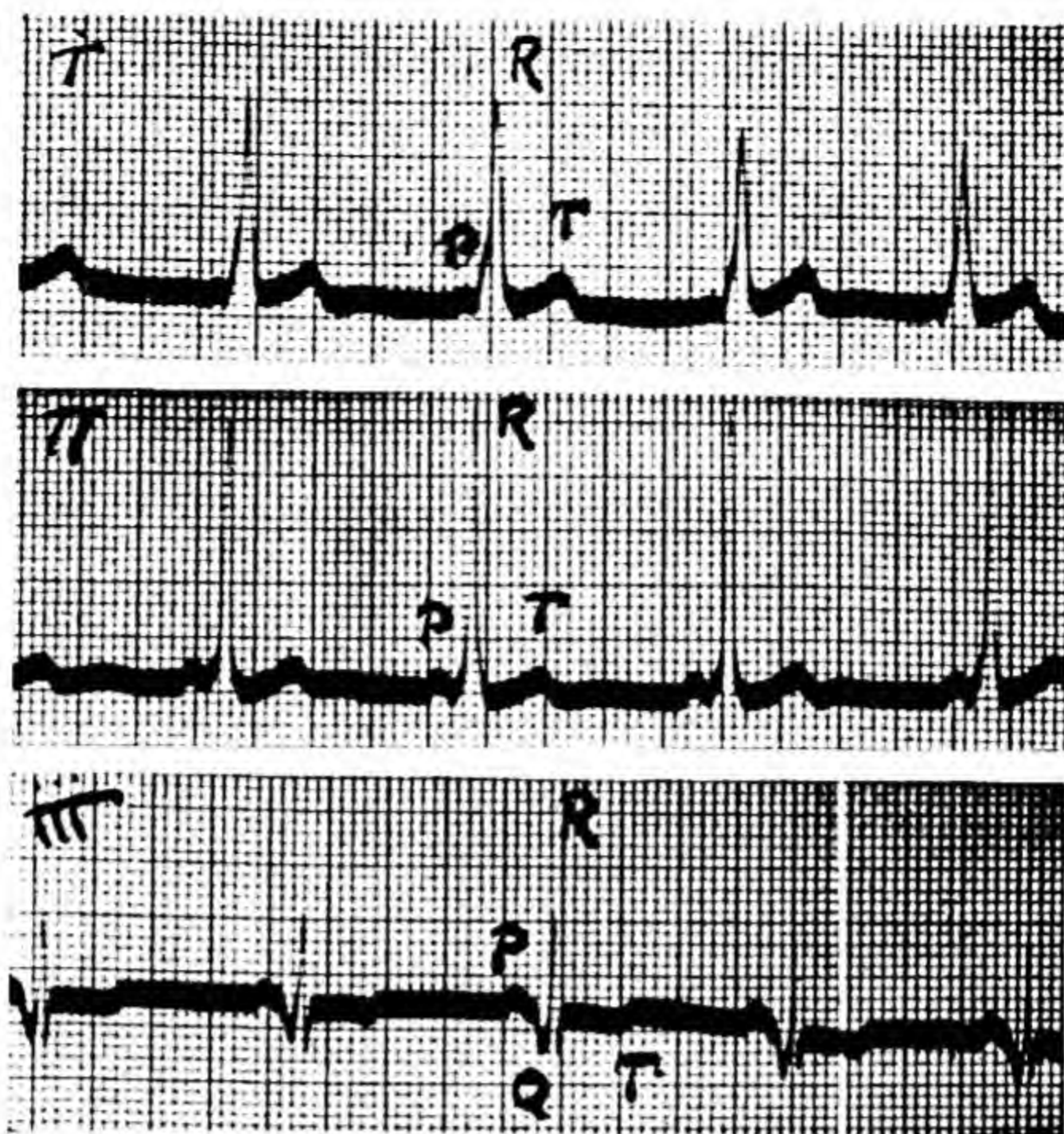


Fig. 131.—Abnormal Form of Ventricular Complex (Wolff-Parkinson-White Syndrome). Note that the P-R interval is very short (0.08 second) and the QRS complex is slurred and broadened (0.11 second). Such peculiar curves are seen in some individuals who have paroxysms of tachycardia but who are otherwise well.

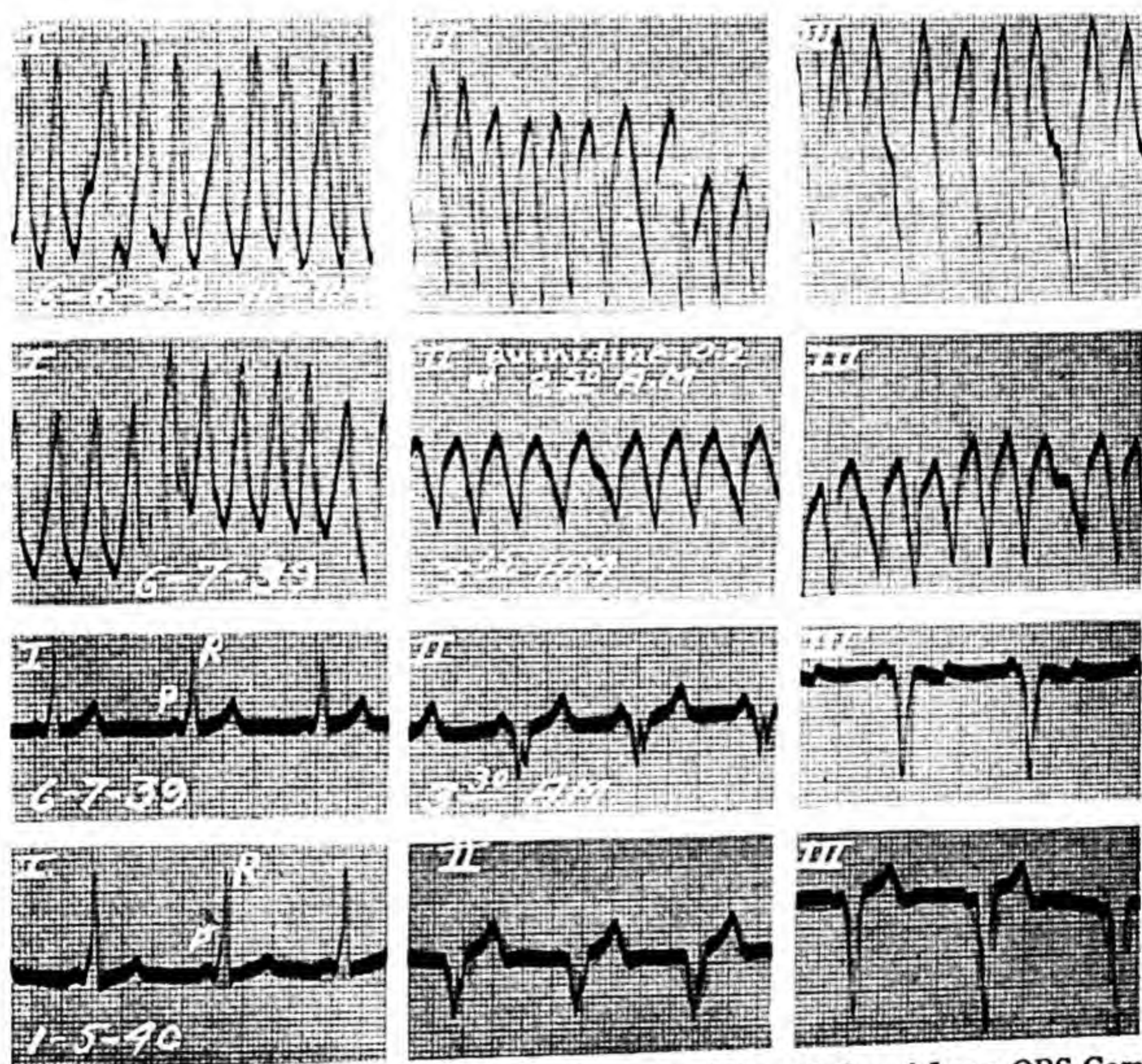


Fig. 132.—Abnormal Form of Ventricular Complex (Short P-R and Long QRS Complex, *i.e.*, Wolff-Parkinson-White Syndrome). The lower two sets of curves show typical short P-R interval (0.1 second) and long QRS interval (0.12 second). The upper two sets show a paroxysm of tachycardia probably ventricular in origin. The heart rate at first was 292; after 0.2 gram of quinidine it slowed to 291 and in less than one hour the rhythm was normal.



intervals are found, a careful inquiry should be made concerning spells of palpitation. It is impossible to recognize this abnormality without electrocardiograms though one might suspect its presence if, in patients subject to attacks of palpitation, one hears an accentuated first heart sound and if conditions such as mitral stenosis, hyperthyroidism, anemia and fever can be eliminated.

**Ventricular Complexes Resulting from Digitalis.**—In any consideration of the significance of an abnormal ventricular complex due regard must be paid to the possible influence of digitalis. This drug produces two main effects. It lengthens the P-R interval and depresses the R-T

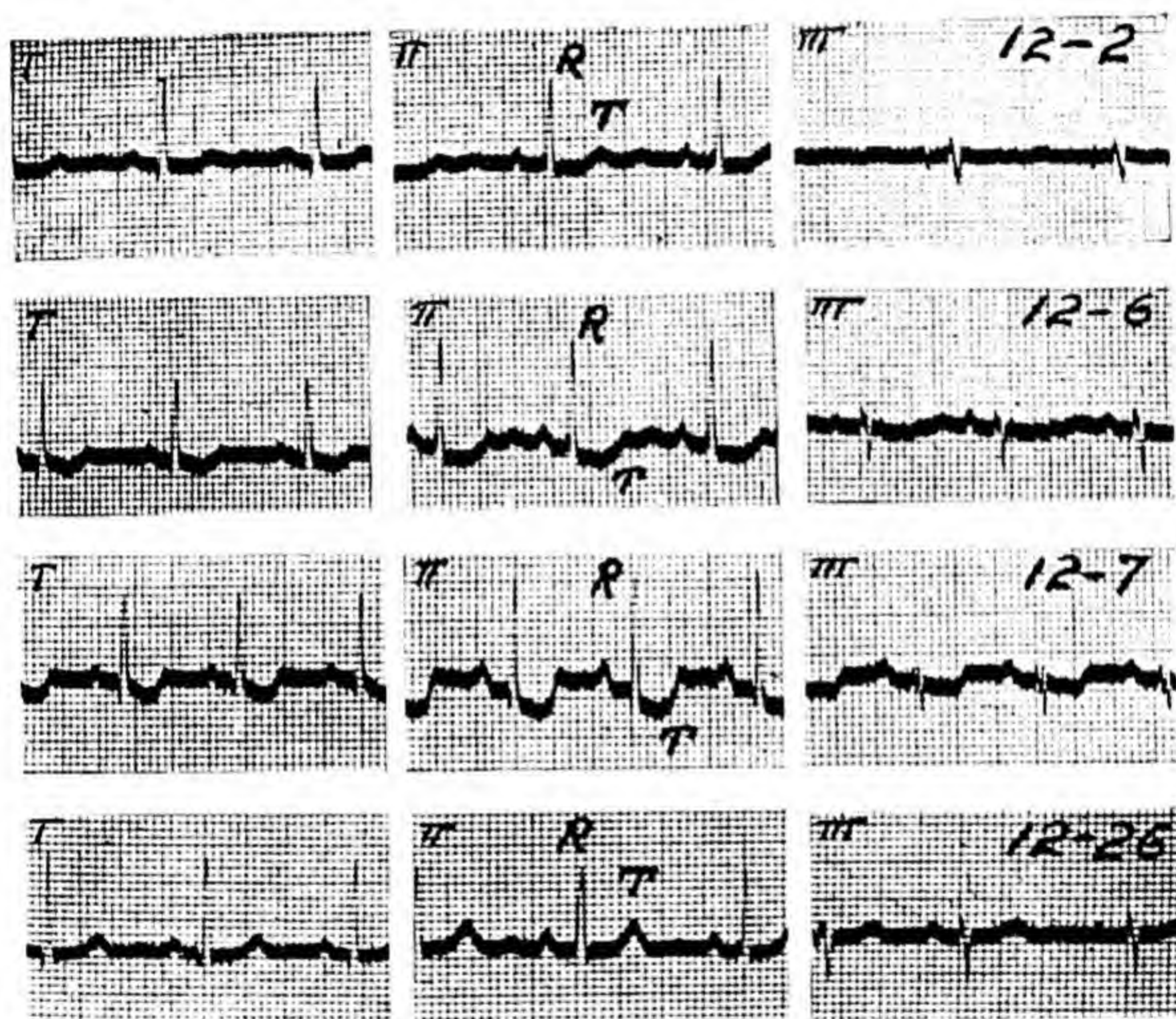


Fig. 133.—Abnormal Form of Ventricular Complex. Digitalis Effect. One and one-half grams of digitalis were given between the first and third tracings. Note the gradual inversion of the T waves in all leads and the return to normal in three weeks. The R-T interval becomes convex downward, unlike the upward convexity in coronary thrombosis. (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

segment. Figure 133 shows the gradual changes in the T wave resulting from a full dose of digitalis. It will be noted that the T wave, particularly in Lead II, gradually becomes depressed and finally inverted. The R-T segment attains a U-shaped inversion with an upward concavity rather than with an upward convexity which characterizes the coronary form of the T wave. Digitalis effects on the T wave are not always seen and do not appear with small doses. They do not represent a toxic action of the drug as they appear when full therapeutic doses are given. It requires two to three weeks for these effects to disappear after the drug is omitted.



## ELECTROCARDIOGRAPHIC INTERPRETATION

Before leaving the subject of electrocardiography, it is necessary to express a word of caution against trying to read too much into the results of this examination. Too often physicians attach significance to minor abnormalities. It must be remembered that variations amongst normal hearts are very great. Slight notching of QRS complexes, differences in amplitude of curves, left and right axis deviation and even flattened diphasic or slightly inverted T waves occur without organic heart disease. Furthermore, when certain curves are diagnostic of myocardial disease they may serve no further purpose in prognosis. In a case of typical acute coronary thrombosis the curves may show very little abnormality or may be returning to a normal configuration and yet the patient may be doing poorly or may suddenly die. It is generally idle to say that "the electrocardiograms are getting better." It is more important to know what the patient is doing clinically than to know what further changes are going on in his tracings. Although, in general, patients with inverted T waves in Lead I have a poor prognosis, there are a great many who live for years and years and in fact there are some who have no clinical evidence of either angina or heart failure. In the extensive use of electrocardiography there is a danger of prostituting the entire subject. Attempts to interpret curves are being made when the leads were applied incorrectly or when the tracings were pasted upside down or wrong end to. Artefacts are being interpreted as pathologic changes and many similar gross errors are being made. Despite all this, with care and intelligence electrocardiography can be a most valuable aid in the treatment of patients with heart disease.

## PULSUS ALTERNANS

There is one disturbance in the mechanism of the heart beat that is truly not within the scope of electrocardiography because it is not diagnosed by this means. However, it needs to be considered for it is important and can easily be recognized. This condition is *pulsus alternans*, by which is meant an alternation in the strength of the beat when the rhythm is regular (Fig. 134). In this condition the impulses of the heart arise regularly in the normal pacemaker and travel through the normal pathways. The electrocardiogram is therefore normal. Every other beat, however, is stronger than the previous one. It must be clearly differentiated from a pseudo-alternation due to prematurity of each second beat. In bigeminy due to extrasystoles every second beat is also small but this results because the contractions occur slightly ahead of time and the ventricles contain less blood. In true alternation the intervals between beats are equal and the weaker contractions are thought to be due to fatigue of the heart muscle or to impaired contractility. It has been hypothesized that isolated bits of ventricular fibers here and there do not enter into systole because of fatigue so that each alternate systole is not as effective as the former one.



Pulsus alternans can be recognized if a pulse tracing is taken from one of the peripheral arteries. It can be felt at the radial pulse where every other beat will be weak. Unless this sign is looked for or unless it is very marked it is generally overlooked. Changes in the volume of the peripheral pulse produced by different phases of respiration often interfere with and obscure the regularity of the alternation. At times the same alternation can be detected in the heart itself by noting an alternation in the intensity of the first heart sound or of an accompanying systolic murmur, or even in the force of the apex impulse. In other words, the alternation may be seen, heard or felt. The simplest and most sensitive way of diagnosing pulsus alternans is with the sphygmomanometer. While taking the blood pressure one should listen carefully and try to detect alternation of the sounds as the pressure falls and is just approaching the systolic level. When the sounds first appear the pressure should be prevented from falling for several seconds. If alternation is present,

*Stevens FZ Nov 5 '19*

*Brachigram.*

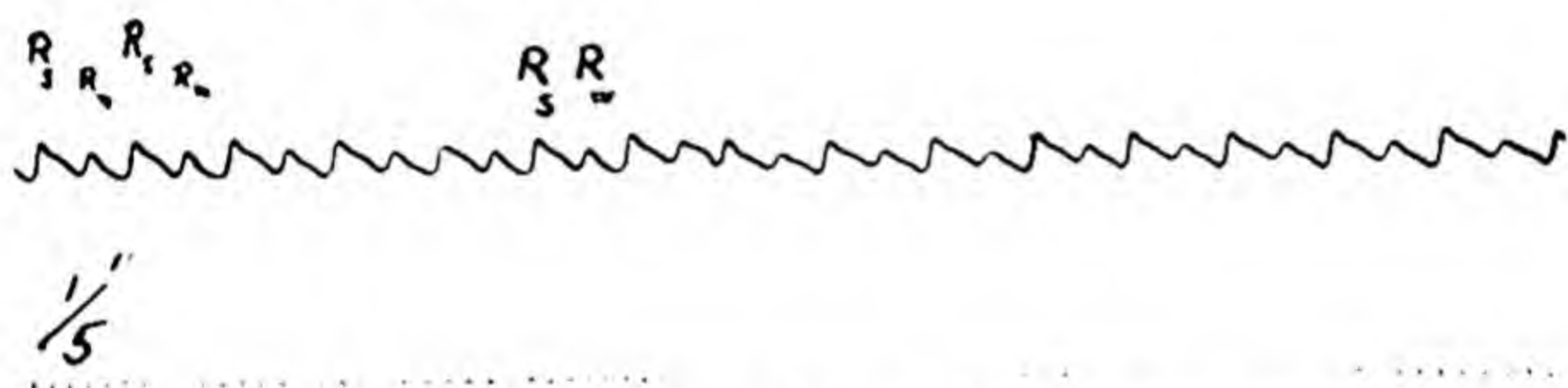


Fig. 134.—Pulsus Alternans. Tracing taken from the brachial artery which shows a regular pulse but waves alternating in strength;  $R_s$  is larger than  $R_w$ . (Author's article in Oxford Loose-Leaf Medicine, vol. II. Courtesy of Oxford University Press.)

the sounds will either alternate in intensity or only the stronger beat will be heard. If the latter takes place, then both sounds will become audible 5 or 10 mm. lower but will still alternate in intensity. This method is more sensitive and just as reliable as the pulse tracing in the diagnosis of pulsus alternans. It should be sought for as a routine procedure in all patients with definite or suspected heart disease.

In some patients pulsus alternans only appears for several seconds after a premature beat and then disappears. Following the compensatory pause the first beat is always exaggerated but now the second beat is markedly diminished, the third is increased and so on for several cycles. The extrasystole brings to the surface the tendency to alternation. This has the same general significance as the more permanent form of alternation. It also may appear during the very rapid rate of paroxysmal tachycardia in a patient without organic heart disease in whom no alternation is present while the rate is slow.

Generally the electrocardiogram fails to show abnormalities that correspond with the pulse abnormalities. Occasionally electrical alternation



takes place, every other QRS complex being large and small. Such alternation is supposed to have the same bearing on prognosis as that of the peripheral pulse. Alternation of the P wave has also been noted during the rapid rate of auricular flutter.

Pulsus alternans occurring in regularly beating hearts, apart from paroxysmal tachycardia, indicates heart muscle disease of a serious degree. It is not seen in normal hearts. It is most common in association with hypertensive heart disease and with disease of the coronary arteries but occasionally is found in valvular cases. It is generally present when the rate is over 90 and may disappear as the rate falls to about 70. In fact, it is very rare when the heart is beating slowly. Its importance lies in its prognostic significance. The length of life after this sign is elicited is on the average not more than a year or two, although there are exceptional patients who carry on quite satisfactorily for more than five years.

### THE PRECORDIAL ELECTROCARDIOGRAM

Until about 1930 clinical electrocardiography was limited to the study of the three conventional limb leads. As a result of the early work of Wilson and his colleagues and that of Wolferth and Wood precordial electrocardiography was introduced into clinical medicine. This has thrown much light on the diagnosis of myocardial disease, especially myocardial infarction, and on the nature of the human electrocardiogram. Because of its recent development, the knowledge derived therefrom has had to be carefully tested and appraised. Conclusions have had to be revised with increasing experience. Just as was the case during the early years with the three conventional leads, it has been necessary to establish the normal variations in the precordial leads and to analyze the circumstances under which the complexes might change and the causes of the various abnormalities encountered. Some of the opinions prevailing at present are still controversial and others that are generally accepted may need revision in the future. However, there is considerable that is already well established and clinically applicable. Before taking up some of the practical applications of precordial electrocardiography, a brief discussion of the theoretical mechanisms involved seems advisable. This has been digested from a recent publication of Wilson and his co-workers.

At the outset one must appreciate some of the differences between registering the electrical potentials of the heart by taking leads from the limbs (the three standard leads) and from the chest wall. The potential variation of the heart beat, *i.e.*, the height of the electrical complex, diminishes rapidly the farther removed from the heart. The potential variation is about thirty times as great over the surface of the ventricles as it is at the limbs. When an electrode is placed over the right arm and on the left leg, it does not matter whether one is an inch higher or lower on the limb as far as the electrocardiogram is concerned. Both points are so far removed from the heart that small changes in the point of contact produce imperceptible changes in the electrical potential. These limb



leads are "bipolar leads." However, when one electrode is placed over the precordium and therefore very close to the heart and coupled with a distant electrode, small changes in position of the former produce marked changes in the form of the electrocardiogram. These are essentially "unipolar leads."

If a small electrode is placed over the surface of the heart and an indifferent electrode on a distant point, and the wires are arranged, as they are in clinical work, so that a positive potential registers an upward deflection, the abrupt downward deflection is called the "intrinsic deflection." The beginning of this deflection is generally the top point of the R wave. This marks the time of activation of the subepicardial muscle in contact with the exploring electrode. The other waves of the QRS complex are due to electrical changes taking place in muscle bundles more removed from the point of contact of the exploring electrode. These are called "extrinsic deflections." Waves that occur before the "intrinsic deflection" are due to excitation of muscle bundles before and those after it due to muscle that was still resting at the time the muscle under the point of contact became active. Between active and resting muscle bundles there is an electromotive force which makes the potential of points towards which the excitation wave is advancing positive, and points which lie behind it negative.

The impulse travels from endocardium to epicardium and through the septum from both sides simultaneously. The potential of the ventricular cavities is negative throughout the QRS interval, unless one side of the septum becomes active before the other as occurs in bundle branch block. If the latter is present, the ventricular cavity activated last (the side that is blocked) will show an initial positivity. If the left branch is blocked most of the septum is activated from the right side towards the left and therefore the left ventricular cavity is positive until the impulse passes through the septum. The electrocardiogram from the outer surface of the delayed ventricle will have two peaks. The first is due to activation of the septum and represents positivity of ventricular cavity which is transmitted through inactive ventricular wall, and the second is due to activation of the ventricular wall adjacent to the electrode. When the delayed impulse reaches the ventricular epicardium (top of second peak) the potential of the electrode on the surface becomes the potential of the ventricular cavity, which is now negative. There follows, therefore, a sharp downward deflection, which is the "intrinsic deflection." If the excitation process is still spreading in some part of the ventricular wall, the negativity of the ventricular cavity outlasts this deflection and an S wave results.

If the subendocardium of some part of the ventricle becomes active before the endocardium just beneath the electrode there is initial negativity instead of positivity and a Q wave results. There can be no Q wave if the cavity is initially positive. This explains why the electrocardiogram of bundle branch block will have no Q wave if an electrode is placed



on the surface of the blocked side, because the cavity is positive early as the impulse travels towards it through the septum from the opposite unblocked side. In general, Q waves are common in those leads in which intrinsic deflections come late in the QRS complex and where R is tall and S is small or absent.

The potential changes over the surface of the heart, which we try to measure by taking chest leads, are somewhat obscured by the potential of the distant point (one of the limbs). For this reason Wilson devised a central terminal connected with three equal resistances of 5000 ohms to each of the three limbs (right arm, left arm and left leg). The potential of such a lead is practically zero throughout the cardiac cycle. When this is coupled with an exploring electrode over the chest the curve reflects the potential of the surface of the heart. This can be more accurately called a uni-polar or semi-direct lead. When such leads are taken they are called  $V_1$ ,  $V_2$ ,  $V_3$ ,  $V_4$ ,  $V_5$ ,  $V_6$ , depending on which position is taken.  $V_1$  is taken from the right costal margin at the fourth space.  $V_2$  is from the left costal margin at the fourth space. The others fall along a broken line drawn from the  $V_2$  position to the apex beat and then horizontally towards the axilla. If the apex beat cannot be found the mid-clavicular line at the fifth interspace is chosen.  $V_3$  is midway between  $V_2$  and the apex impulse.  $V_4$  is at the mid-clavicular line.  $V_5$  is in the anterior axillary line and  $V_6$  is at the mid-axillary line. When precordial leads are taken with an ordinary electrode placed in these various positions and coupled with the left leg they are called  $CF_1$ ,  $CF_2$ , et cetera; if the indifferent electrode is on the right arm it is  $CR_1$ ,  $CR_2$ , et cetera; if the left arm or back is used the terms employed are  $CL_1$ ,  $CL_2$ , et cetera or  $CB_1$ ,  $CB_2$ , et cetera.

The electrocardiograms obtained using a central terminal electrode as has just been described are slightly different from those obtained if a precordial electrode is coupled with one of the limbs. They are also different if the exploring electrode is coupled with one limb as compared with another limb. The observations to be discussed in the following paragraphs are applicable for the most part to all the different methods. Although the most common practice has been to use the left leg for the indifferent electrode, it seems more logical to use the central terminal and this may well become the accepted method in the future.

**Limb Potentials.**—By coupling the central terminal with an electrode on any of the limbs the potential of one of the arms or legs can be determined. These are indicated by the expressions  $V_R$ ,  $V_L$  and  $V_F$  for the right arm, left arm and left leg respectively. Other notations may be made, such as  $V_E$  for a point over the ensiform. The following generalizations are applicable to the form of these unipolar limb leads. When the standard Leads I and II are similar  $V_R$  resembles these leads upside down. When Lead III is the inverse of Lead I,  $V_2$  is like Lead I, and when Leads II and III are alike  $V_F$  has a similar form.

**Position of the Heart from Electrocardiographic Standpoint.**—A study



of the electrical potential of the individual limbs,  $V_R$ ,  $V_L$ ,  $V_F$ , as compared to the standard three leads has led to some new concepts concerning the electrical axis of the heart amongst normal individuals and has thrown light on methods of differentiating such normal variations from those produced by hypertrophy of either ventricle. Normally the heart may be regarded as being in one of six positions, depending on individual characteristics such as the shape of the chest or height of the diaphragm. The following method enables one to identify these types.

Vertical position:  $V_L$  resembles  $V_1$  and  $V_2$ ;  $V_F$  resembles  $V_5$  and  $V_6$ .

Semivertical position:  $V_F$  resembles  $V_5$  and  $V_6$ ; QRS of  $V_L$  is small.

Intermediate position:  $V_L$  and  $V_F$  are similar and resemble  $V_5$  and  $V_6$ .

Semihorizontal position:  $V_L$  resembles  $V_5$  and  $V_6$ ; QRS of  $V_F$  is small.

Horizontal position:  $V_L$  resembles  $V_5$  and  $V_6$ ;  $V_F$  resembles  $V_1$  and  $V_2$ .

Indeterminate position: No relation between limb and precordial leads.

**Normal Precordial Leads.**—When the electrode is placed over the right side of the precordium ( $V_1$ ,  $V_2$  or  $CF_1$  and  $CF_2$ ) the R wave is small, narrow, and comes early, and S is deep and broader. Over the left side ( $V_5$  and  $V_6$ ) R is tall, relatively wide and often is preceded by a small Q and followed by an S. The peak of R (the onset of the intrinsic deflection) is 0.02 second later over the left side than the peak of R on the right side of the precordium, because the left ventricle is thicker than the right. The intermediate zone  $V_3$  and  $V_4$  has characteristics between these two types and often displays notching. Q waves are practically never seen in  $V_1$  or  $V_2$  and often are absent in all precordial leads. Small Q waves (1 to 3 millimeters in depth), however, may be present normally in  $V_3$  to  $V_6$ . R waves should increase in size from  $V_1$  to  $V_4$  and then decrease beyond. S waves may or may not be present in any of the positions. They are greatest at  $V_1$  and  $V_2$  and then slowly decrease as one proceeds towards the left axilla.

Except for position  $V_1$ , all T waves are normally upright in precordial leads. At  $V_1$  the T waves may be upright or inverted. This may prove to be also true in a rare case with  $V_2$ . However, inversion of the T waves in the other position does not necessarily mean myocardial infarction, although it often does. Hypertrophy of the right ventricle can produce inverted T waves at  $V_1$  and  $V_2$  and hypertrophy of the left ventricle may produce inversion of T waves at  $V_5$ ,  $V_6$  and possibly  $V_4$ . Apart from the peculiar form of the inverted T waves, the inversion that accompanies ventricular hypertrophy is apt to be associated with QRS complexes that are somewhat broad and that have a large area, while those indicative of myocardial infarction have sharper QRS complexes with a smaller cross area.

Finally the S-T segment is normally iso-electric or only slightly elevated or depressed. A marked deviation of the S-T segment is often but not always indicative of an acute myocardial infarction. For some unknown reason a marked elevation of the S-T segment may rarely persist



for many months after the acute phases of myocardial infarction. Occasionally an elevation of one millimeter above the base line may be significant and at other times such a change may need to be disregarded or appraised only in relation to other changes in the tracings.

With the foregoing general review in mind let us now see how one may apply such knowledge in actual clinical problems. The main purpose of precordial electrocardiography is to obtain aid in the diagnosis of myocardial infarction and the study of coronary or myocardial disease. The arrhythmias are generally better studied by the standard three leads, although occasionally auricular complexes are more easily analyzed by leads taken directly from the chest or from the esophagus.

Figure 135 shows the standard three leads and the six chest leads ( $CF_1$  to  $CF_6$ ) of a normal man twenty-six years old. The T waves are all upright though  $CF_1$  might have shown an inverted T. The R wave

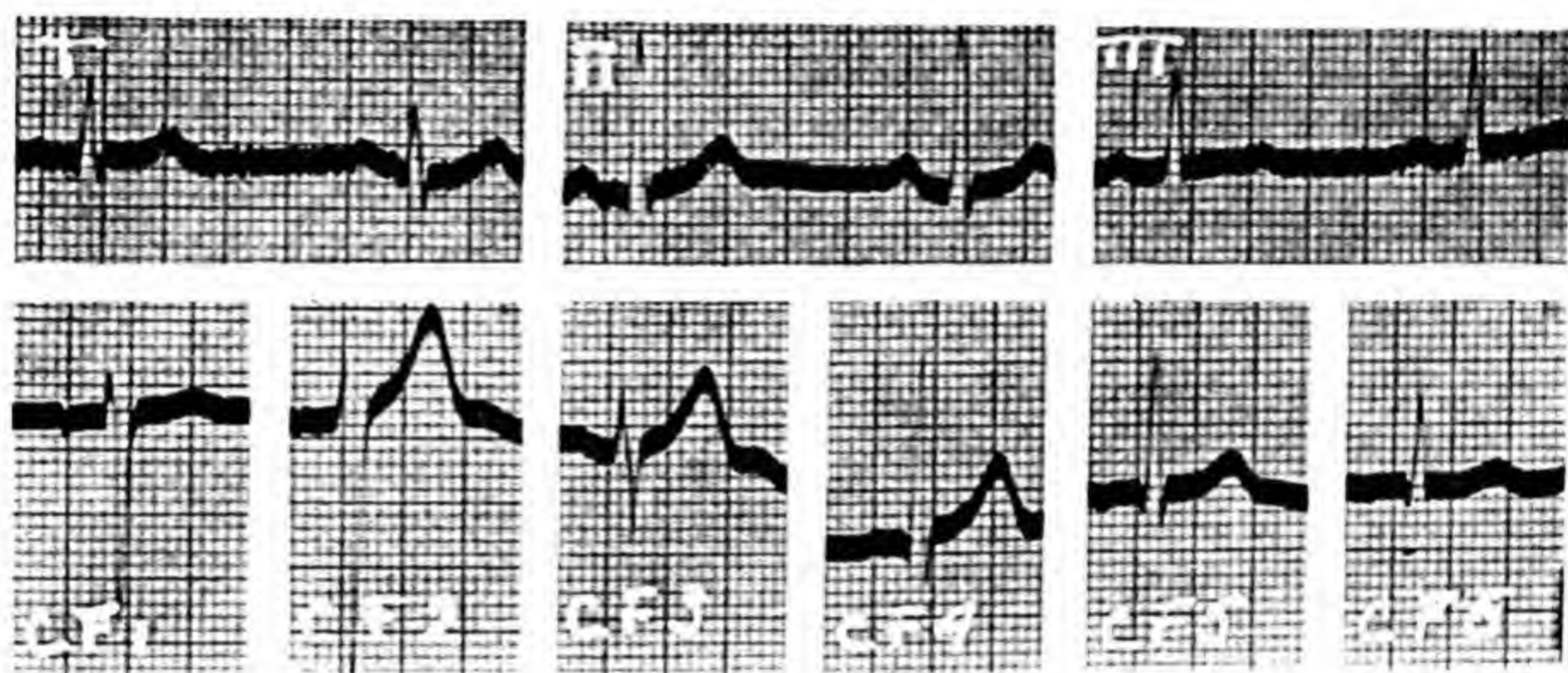


Fig. 135.—Normal Electrocardiogram—Six Precordial Leads. The upper strip shows the three standard leads, which are normal. The lower strip shows the leads from six points over the precordium ( $CF_1$  to  $CF_6$ ). Note that the T waves are all upright and that an R wave is present in all leads; normally  $CF_1$  may show an inverted T and an absent R. Very slight deviation of S-T may be present normally (see  $CF_4$ ). The patient was a normal male twenty-six years old.

gradually increases in size from  $CF_1$  to  $CF_4$  and then decreases. S waves are very large over the right side of the precordium and become steadily smaller on approaching the left axilla. The S-T junction is slightly but definitely elevated in  $CF_4$ , but the general contour of the complexes is normal. Figure 136 illustrates similar peculiarities in a man forty-eight years old, who had no heart disease. Here the central terminal electrode was used and unipolar leads were obtained from the three extremities ( $V_R$ ,  $V_L$ ,  $V_F$ ) as well as the six precordial leads ( $V_1$  to  $V_6$ ). By referring to the previous discussion of unipolar leads it follows that this heart was in the vertical position.

Figures 137 and 138 are instances of left and right axis deviation. The former is from a stocky man with no evidence of heart disease. The left axis deviation here is the result of the heart lying in a semi-horizontal position. The other is from a case of mitral stenosis. The unipolar limb leads indicate that the heart lies in a semi-vertical position. Different



positions of the heart may be detected by comparing the form of the complexes in the unipolar leads with those in the precordial lead according to the method discussed in a previous paragraph.

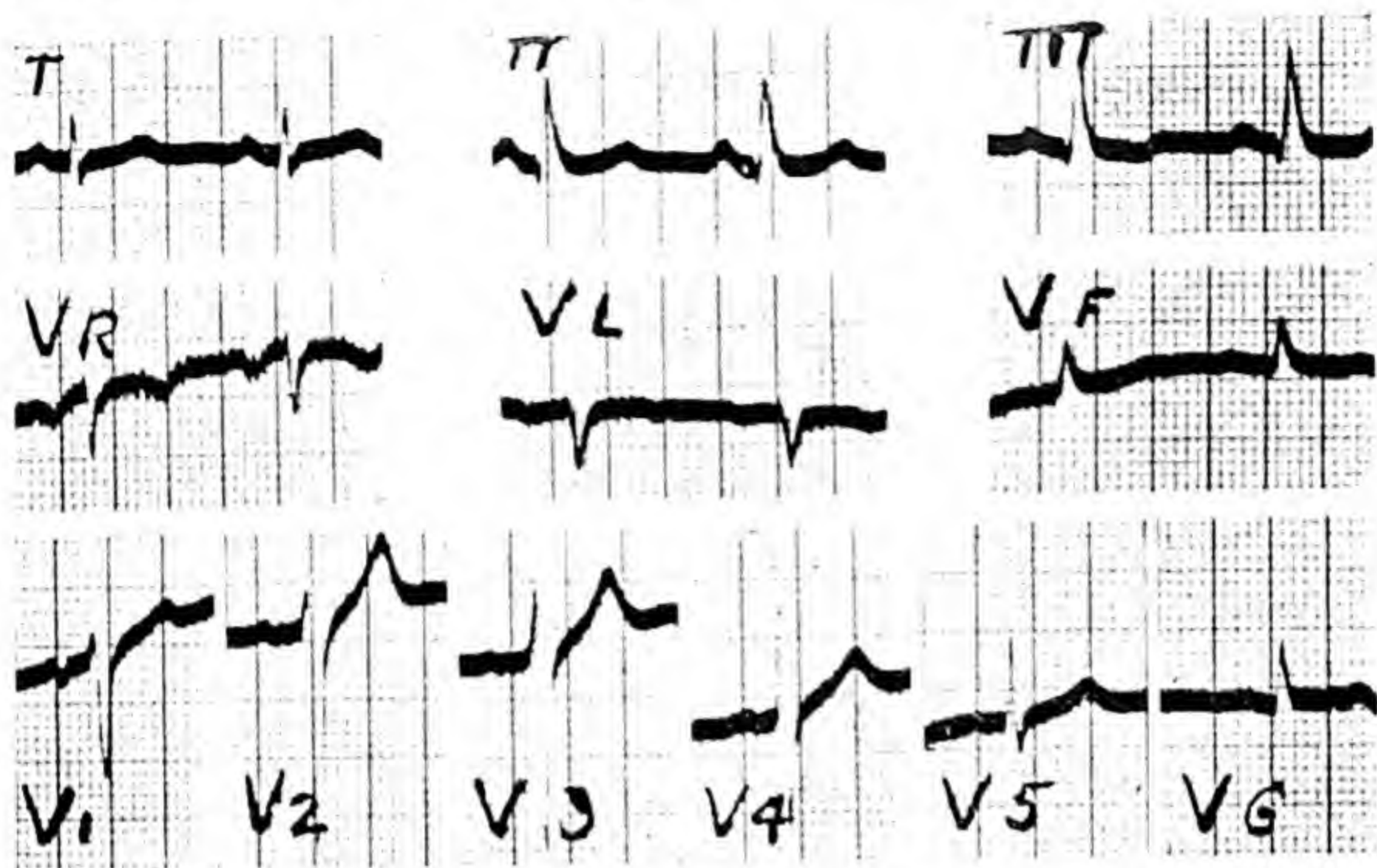


Fig. 136.—Normal Electrocardiograms—Three Unipolar Limb Leads and Six Precordial Leads. The upper set shows three standard leads. The middle set shows three unipolar limb leads,  $V_R$  (right arm),  $V_L$  (left arm),  $V_F$  (left leg). The lowest set shows leads from the six precordial positions. The position of the heart is vertical because  $V_L$  resembles  $V_1$  and  $V_2$  and  $V_F$  resembles  $V_5$  and  $V_6$ .

**Left Bundle Branch Block.**—When the standard electrocardiograms are quite typical of block of one or the other types of bundle branch block, precordial leads are not needed for diagnosis. When there is any

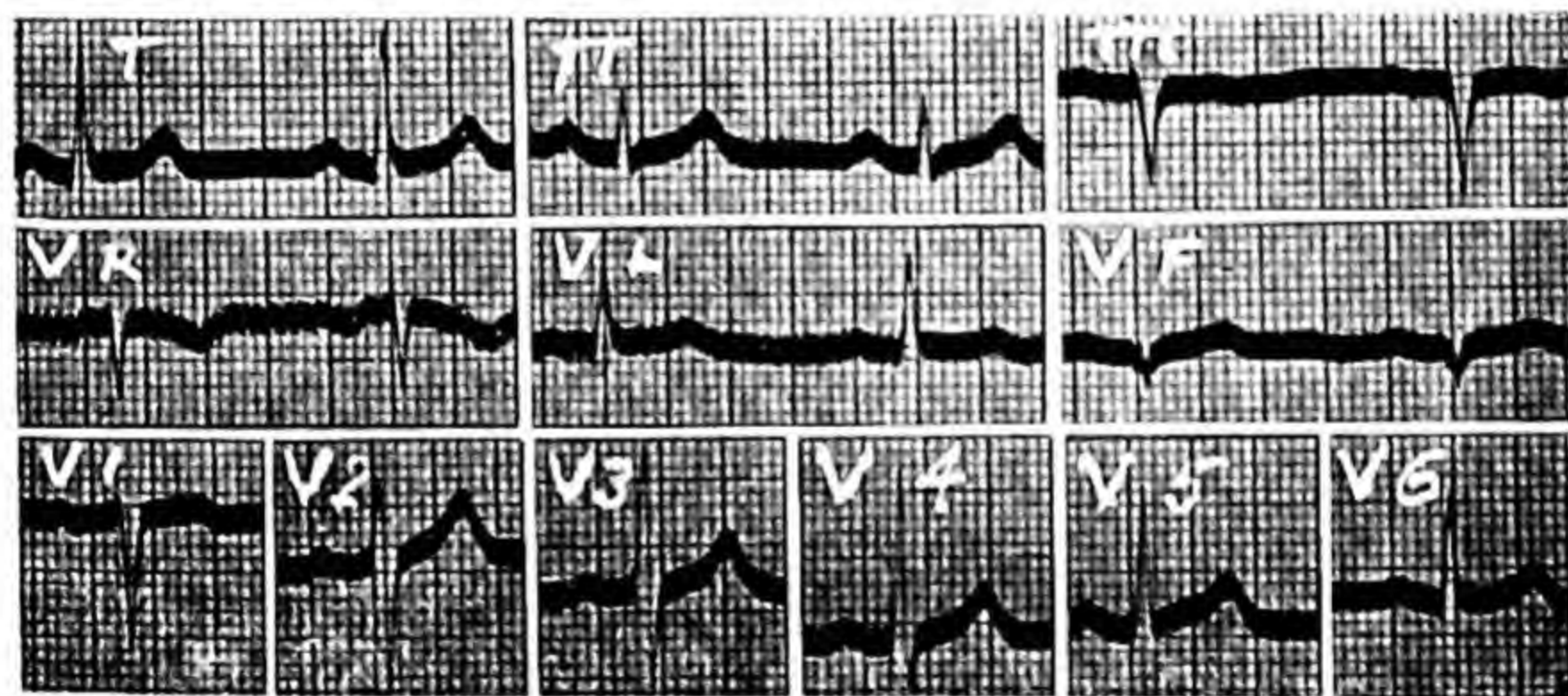


Fig. 137.—Left Axis Deviation—Normal Heart. Upper set shows left axis deviation in the three standard leads. The middle set shows the three unipolar limb leads ( $V_R$ ,  $V_L$ ,  $V_F$ ). The lowest set shows the six precordial leads ( $V_1$  to  $V_6$ ). The heart is in the semi-horizontal position because  $V_L$  resembles  $V_5$  and  $V_6$ , and QRS of  $V_F$  is small. The patient was a man fifty-five years old of a stocky build but having no heart disease.

doubt, however, a series of tracings from the six positions across the chest may supply convincing data upon which to make an accurate diagnosis. If the block is in the left branch, the impulse reaching the left ventricular surface is much delayed and therefore the "intrinsic deflec-



tion," the beginning of which represents the arrival of that impulse at the ventricular epicardium, will occur late after the onset of the QRS

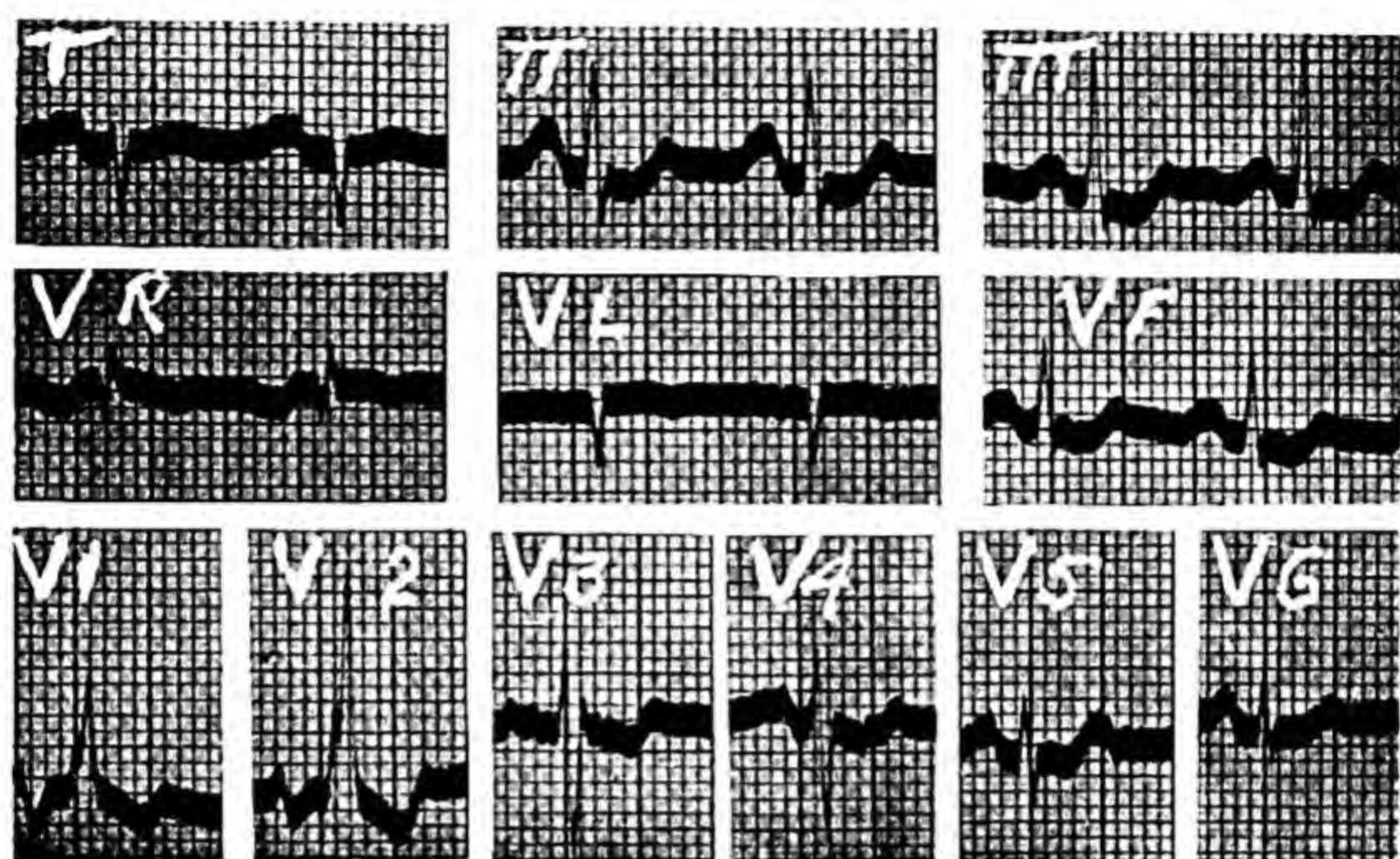


Fig. 138.—Right Axis Deviation—Mitral Stenosis. The upper set shows right axis deviation in the three standard leads. The middle set shows the three unipolar leads ( $V_R$ ,  $V_L$ ,  $V_F$ ). The lowest set shows the six precordial leads ( $V_1$  to  $V_6$ ). The heart is in semi-vertical position because  $V_F$  resembles  $V_6$  and  $V_6$  and QRS of  $V_L$  is small. The patient was a thin woman, thirty-six years of age, with definite signs of mitral stenosis.

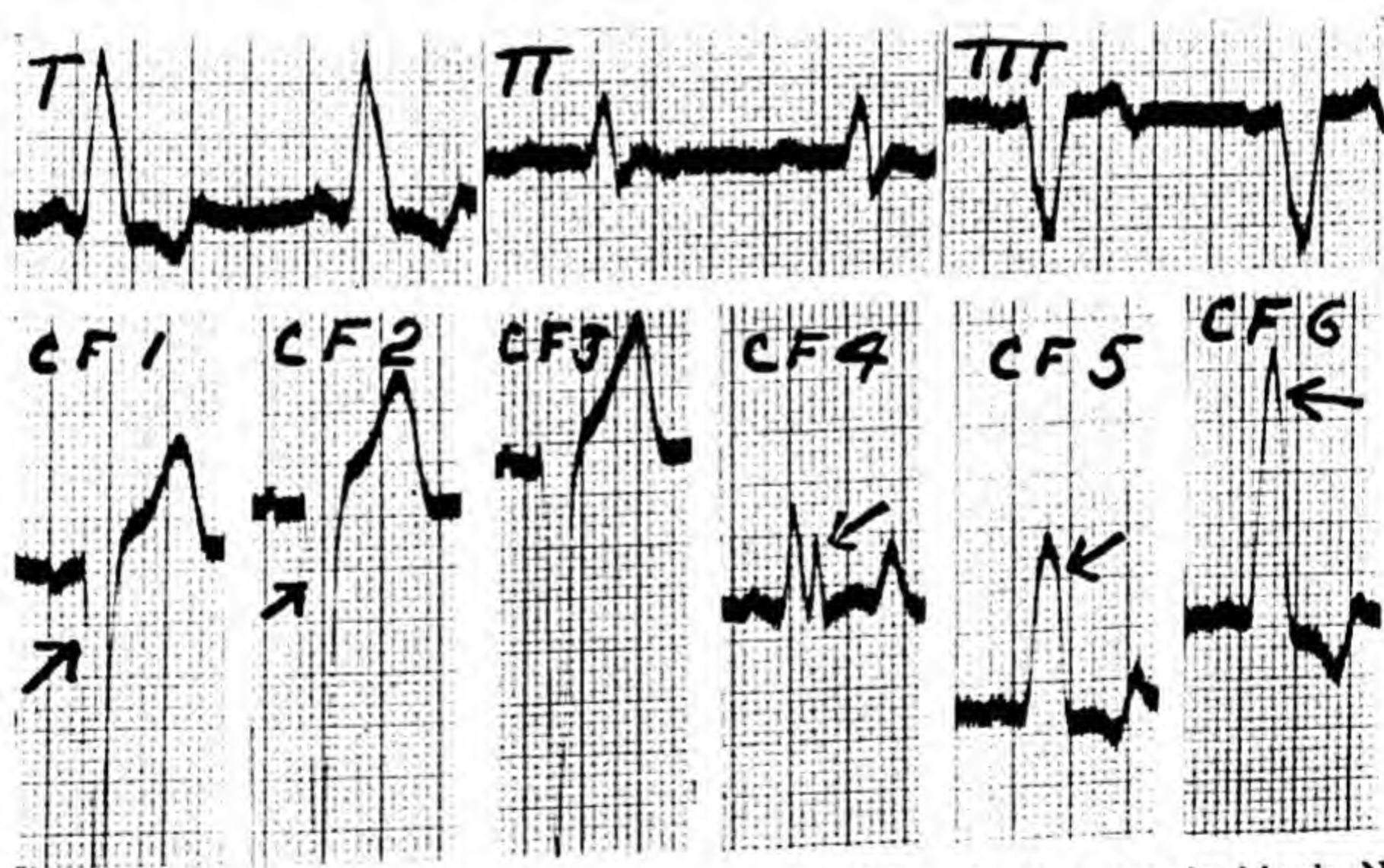


Fig. 139.—Left Bundle Branch Block. The upper set shows the three standard leads. Note that the QRS measures 0.15 second and there is no  $S_1$ . The lower set shows the six precordial leads from the chest and left leg,  $CF_1$  to  $CF_6$ . Note that the sharp downward stroke or intrinsic deflection (see arrow) comes early, over the right ventricle ( $CF_1$ ,  $CF_2$  and  $CF_3$ ) and very late over the left ventricle ( $CF_4$ ,  $CF_5$  and  $CF_6$ ). The latter QRS complexes often show coarse splitting of the waves as in  $CF_4$ . These curves denote delay or block in the left ventricle. The patient was a man, fifty-three years of age, who had a coronary thrombosis two years before.

complex in the leads over the left ventricle ( $V_5$  and  $V_6$  and possibly  $V_4$ ). Contrariwise the intrinsic deflection over the right or unblocked ventricle ( $V_1$  and  $V_2$ ) will occur early in the QRS cycle (Fig. 139). Further-



more, there can be no Q wave in  $V_5$  or  $V_6$  when there is left bundle branch block because the left ventricular cavity is positive early in the cycle. At times marked left ventricular hypertrophy may produce waves that resemble those of left bundle branch block, but the former rarely will display a QRS interval as long as 0.12 second, and will not show such a tardiness of the intrinsic deflections in  $V_5$  or  $V_6$  and may show a Q wave in these positions.

**Right Bundle Branch Block.**—When the right branch of the bundle of His is blocked the standard leads often reveal fairly characteristic electrocardiograms. The QRS complex measures 0.12 second or more and there is a broad  $S_1$ . However, when there is doubt as to the diagnosis, precordial leads can similarly offer convincing evidence for or against the diagnosis. In right bundle branch block the delay is in the right ventricle and the intrinsic deflection in positions  $V_1$  and  $V_2$  will come late

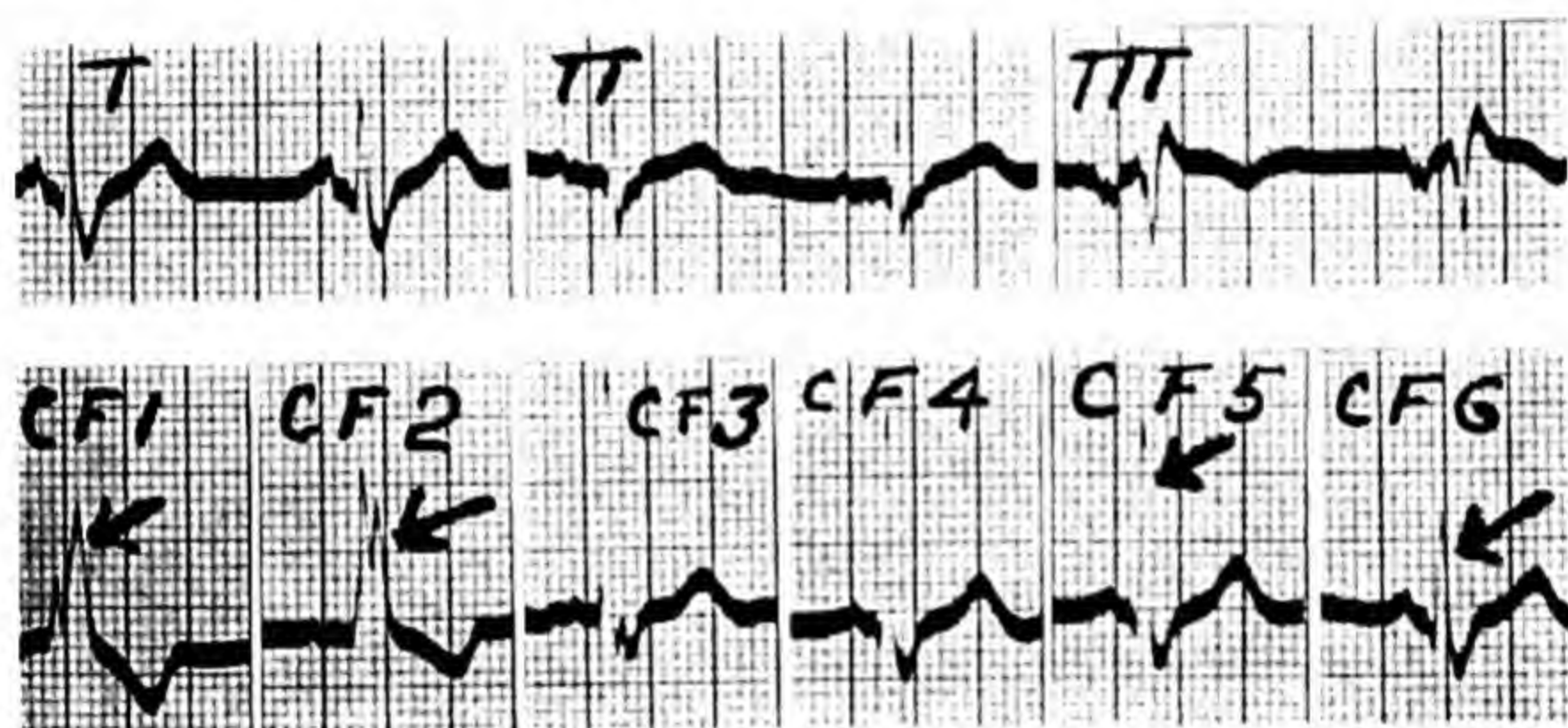


Fig. 140.—Right Bundle Branch Block. The upper set shows three standard leads. Note that  $S_1$  is broad and QRS = 0.13 second. The lower set shows the six precordial leads from the chest and left leg,  $CF_1$ ,  $CF_2$ – $CF_6$ . Note that the last sharp, downward stroke of the R wave or intrinsic deflection (see arrow) comes late in the QRS cycle in  $CF_1$  and  $CF_2$  (over the right ventricle) and comes early in  $CF_5$  and  $CF_6$  (over the left ventricle). This denotes that the block or delay is in the right ventricle. The patient was a woman, forty-eight years of age, with rheumatic aortic stenosis.

after the onset of the QRS complex, because the electrode then lies over the right ventricle, and it will come early in positions  $V_5$  and  $V_6$ , where it lies over the unblocked left ventricle (see Fig. 140). In considering the time of occurrence of the intrinsic deflection due regard must be had for the thickness of the ventricles. There is some delay because of hypertrophy itself, but never as much as results from bundle branch block.

**Myocardial Infarction.**—The main contribution that precordial electrocardiography has made to clinical medicine is in the diagnosis of myocardial infarction. In most cases the three standard leads, or a series of such leads taken on different days, are diagnostic. In some cases, however, the precordial leads may be very distinctive when the conventional leads are quite normal or equivocal. This is true both for the acute stage and the chronic or healed stage. It has also become clear that one precordial lead may at times be entirely inadequate when the exploration



of several points over the chest may reveal diagnostic data of importance. Although there is still some difference of opinion as to the most useful application for the indifferent electrode (left leg, left arm, right arm, or back) this is much less important than the need for various positions for the exploring electrode. It is not unlikely that eventually the central terminal of Wilson will be used for precordial electrocardiography, thereby doing away with all differences in technique concerning the indifferent electrode.

For practical purposes one precordial lead, *e.g.*,  $CF_4$ , may be taken as a routine, and if the diagnosis of myocardial infarction is still in doubt or if the question of bundle branch block is involved a series of three or six chest leads should be taken. During the acute stage only one or two of the various chest leads may show the elevation or depression of the S-T segment or the peculiar rounding and sharp late inversion of the T wave. Similarly the R wave may disappear or be preceded by a large

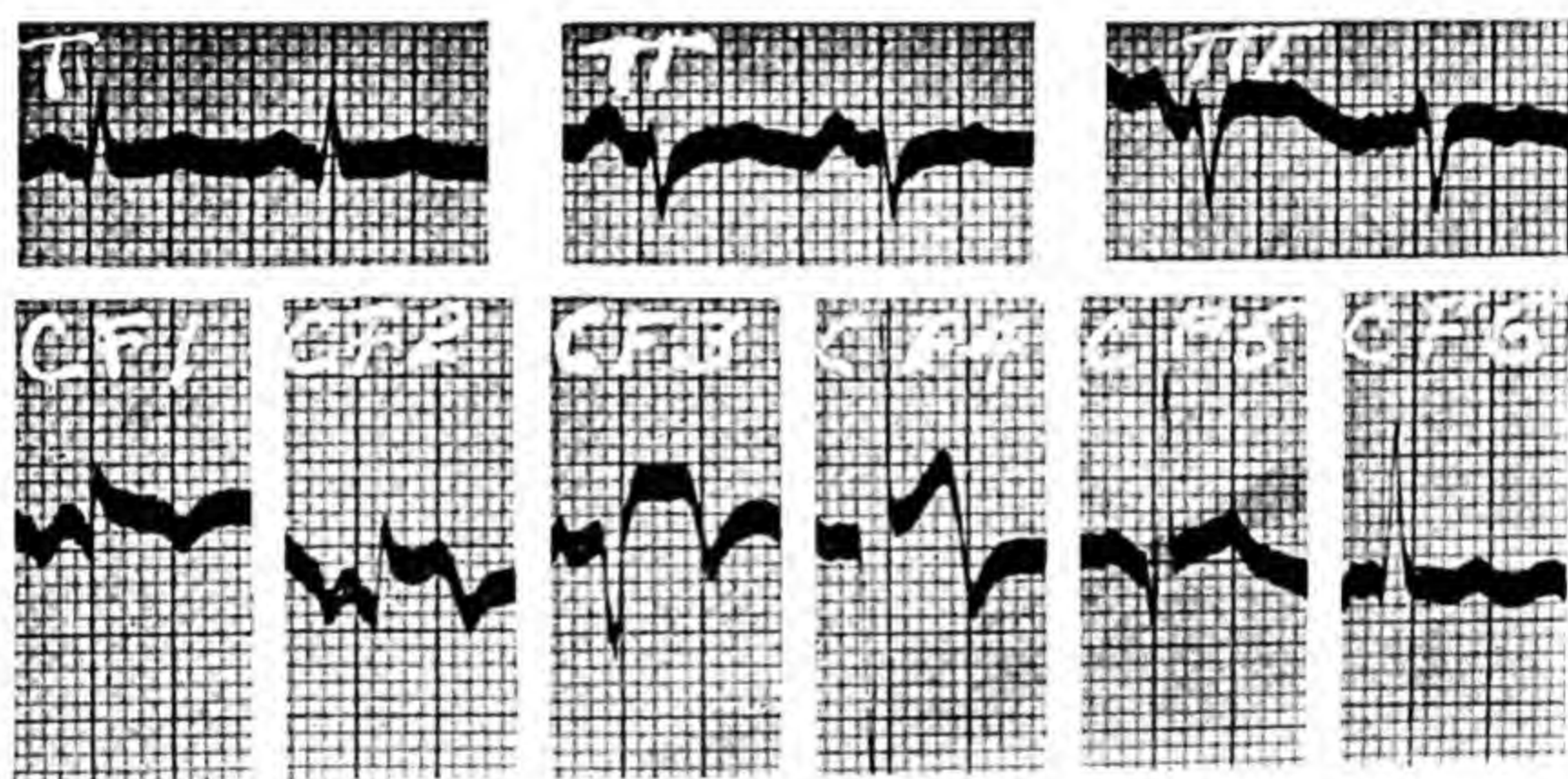


Fig. 141.—Precordial Leads of Acute Anterior Myocardial Infarction. The upper set shows three standard leads that are not definitely diagnostic of infarction. The lower set of six precordial leads shows absence of initial upward deflection in  $CF_1$  to  $CF_3$  and an elevation of S-T in  $CF_3$  to  $CF_6$  with late inversion of T waves. These are diagnostic of anterior infarction. The patient was a man seventy-five years old with a known old gastric ulcer, who developed pain in the xiphoid region, which was first misinterpreted as due to a subacute perforation of the stomach.

Q wave in only one or two of the various leads. This also is indicative of an area of infarction in the anterior part of the heart. The number of positions across the precordium in which these various signs will be present will depend on the size and location of the area involved. It must be recalled that chest leads are mainly helpful in detecting anterior or lateral infarctions although at times the acute stages of posterior infarctions may also be recognized by a marked depression of the S-T segment.

Figure 141 is an instance in which the precordial leads gave conclusive evidence of an acute anterior myocardial infarction when the diagnosis could not be definitely made from the standard leads. The tracings show an initial downward deflection in  $CF_1$  to  $CF_3$  and marked elevation of S-T segment with late inversion of T in  $CF_3$  to  $CF_6$ . These curves are



quite diagnostic of acute myocardial infarction. In this case the electrocardiographic diagnosis was most valuable as the patient was known to have had an ulcer of the stomach and the pain in the xiphoid region was first misinterpreted as being due to a subacute perforation of the stomach.



Fig. 142.—Acute Coronary Thombosis with Anterior Myocardial Infarction. This series of tracings shows three standard leads and  $CF_4$  beginning about four hours after the onset. The standard leads go through the customary changes of an anterior infarction, *i.e.*, elevation of  $S-T_1$ , finally becoming an inverted  $T_1$  and a depressed  $S-T_2$ , becoming a sharply upright  $T_2$ . Note that the first set shows a very high take off of  $S-T_4$ , which remains elevated for over a week and finally becomes sharply inverted. The  $R_4$  is absent throughout. This is characteristic of anterior myocardial infarction. The lowest set shows the six precordial leads,  $CF_1$  to  $CF_6$ . Note that  $R$  is absent and  $T$  is sharply inverted over the first four positions, indicating a large area of infarction. The patient was a man forty-one years old, who recovered and became symptomless.

Figure 142 is another instance of acute anterior infarction showing the progressive changes in lead  $CF_4$  from the very high elevation of  $S-T_4$  taken several hours after the onset of the attack to the sharp marked inversion nineteen days later. In this case the standard leads were quite diagnostic. The last tracing shows the six precordial leads, which indicate an extensive area of involvement as  $R$  is absent and  $T$  is sharply inverted in position  $CF_1$  to  $CF_4$ . Such curves may continue unchanged



indefinitely or in the course of time the T waves may gradually return to a more normal form. Only rarely do the QRS changes disappear.

Similar electrocardiographic changes in the precordial leads are shown in Figure 143. Here also the initial upward deflection is lacking in  $V_1$  to  $V_3$  and the T waves are abnormal. It is of some interest that twenty-two days after the first set of tracings were made, the R wave reappears in  $V_4$ , although the T waves remain inverted. The unipolar leads indi-

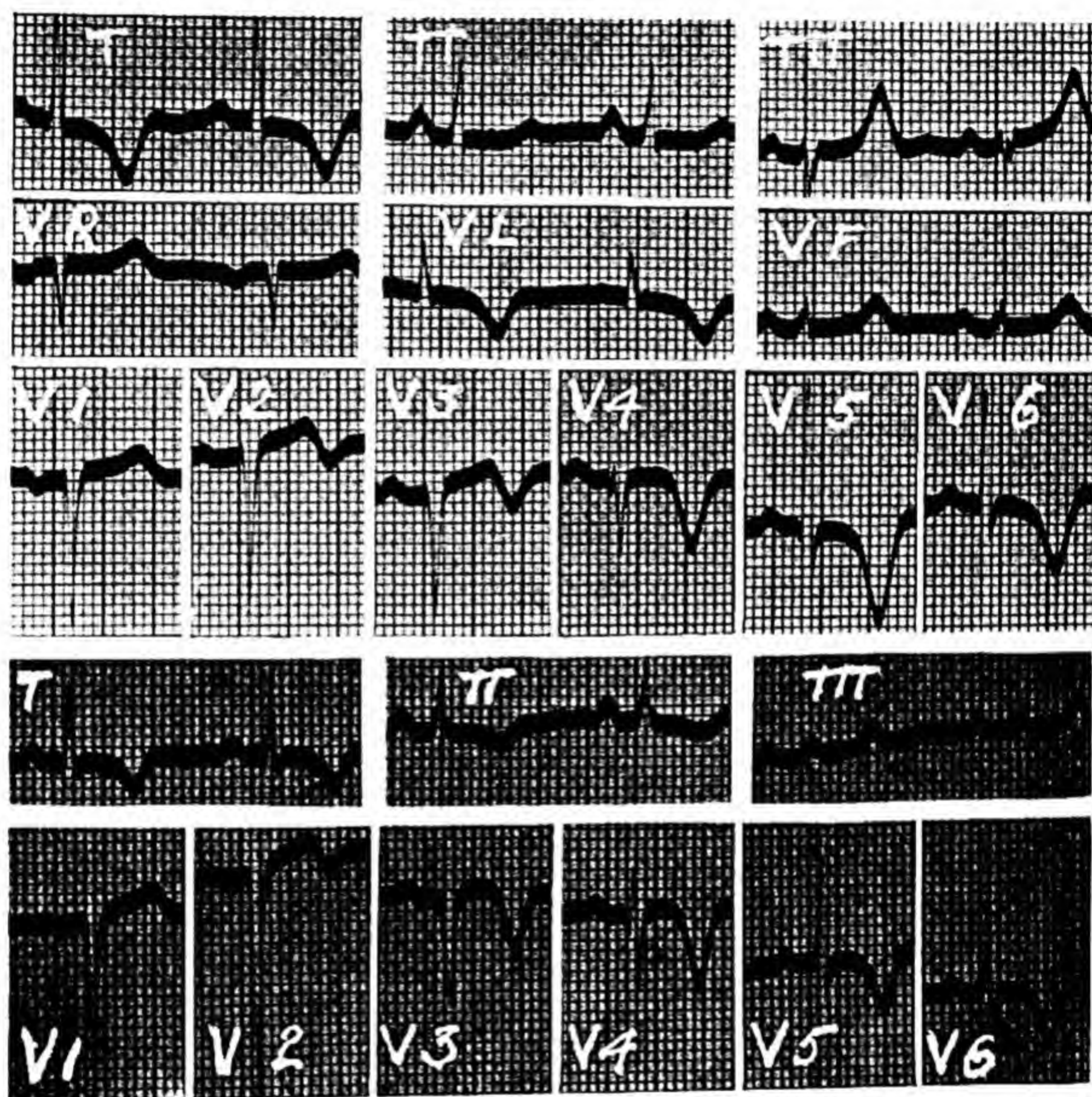


Fig. 143.—Precordial Leads of Acute Anterior Myocardial Infarction. The upper three strips taken October 5, 1941, show the three standard leads, the three unipolar limb leads ( $V_R$ ,  $V_L$ ,  $V_F$ ) and the six precordial leads ( $V_1$  to  $V_6$ ). Note the absence of R waves in  $V_1$  to  $V_3$  and the gradual appearance of dipping and inversion of T from  $V_2$  to  $V_6$ . The lower two strips were made October 27, 1941, and show slight regression of the abnormalities. The patient was a woman, fifty-three years of age, who had a coronary thrombosis two months before and another twelve hours before the first tracing was made. Recovery was satisfactory.

cate that the heart was in a semi-horizontal position because  $V_L$  resembles  $V_5$  and  $V_6$ , and the QRS of  $V_6$  is small.

It has been stated that normally the R waves increase in height in the precordial leads from  $CF_1$  or  $V_1$  to  $CF_4$  or  $V_4$ . When the reverse occurs and the height decreases, especially if it becomes absent in positions  $CF_3$  or  $CF_4$ , the evidence points strongly to anterior infarction. This is illustrated in Figure 144.

Ordinarily, when left bundle branch block is present and an infarction



occurs in the left ventricle, electrocardiograms fail to show the evidence of myocardial infarction. An exception to the general rule is shown in Figure 145. Here a series of tracings reveal definite characteristic changes. The bundle branch block was also transient so that curves could be studied in block and out of block. Both the early and late abnormalities of muscle injury are apparent.

**Esophageal Leads.**—It is evident from the preceding discussion that precordial electrocardiography is often very valuable in the diagnosis of anterior infarction. When the lesion is on the posterior aspect of the left ventricle changes that are diagnostic are generally lacking because the electrode is too far removed from the area of injury. During the acute stages, not infrequently, a marked depression of the S-T segment in some of the precordial leads may be found which is quite diagnostic, but

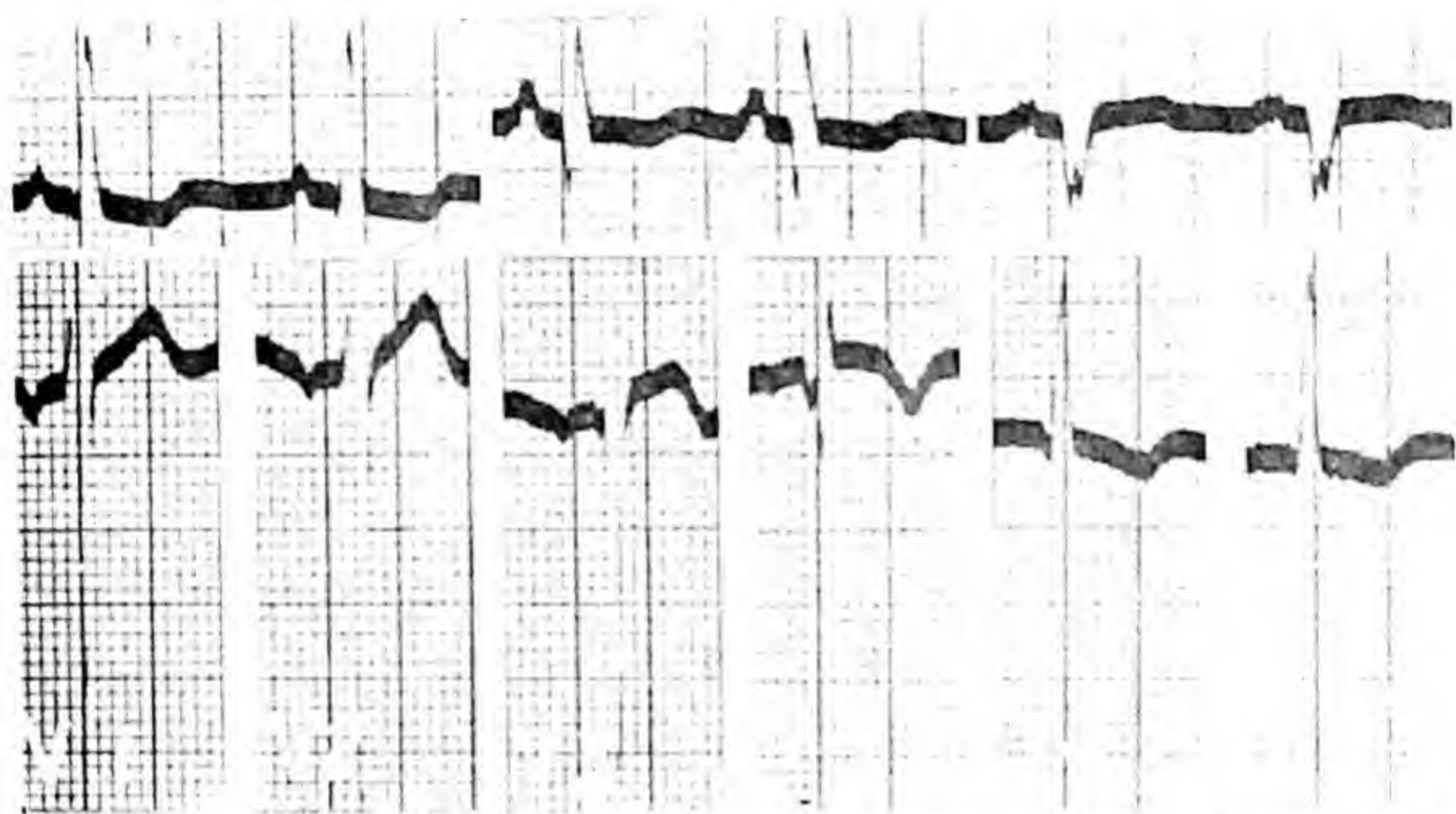


Fig. 144.—Precordial Leads of Old Anterior Myocardial Infarction. The upper set shows the three standard leads and the lower the six precordial leads taken February 16, 1948. Note that the R wave gradually decreases in size from  $V_1$  to  $V_6$  so that it is absent at  $V_1$  and  $V_4$ . There are also changes in T in  $V_4$  to  $V_6$ . There may have been an old posterior lesion as well. The patient was a man thirty-nine years old with severe chronic arthritis, who had an acute coronary thrombosis in April of 1942.

with recovery nothing abnormal may remain except an unusually large upright T wave. Information to be derived from QRS changes, which are so valuable in anterior lesions, is entirely lacking. If an electrode could be applied close to the posterior portion of the heart, significant changes would be detected in a similar fashion as are obtained in an anterior lesion when electrodes are placed over the precordium. This can be done by feeding a special type of electrode through the nose, down the esophagus until the tip reaches the area behind the heart. This electrode is then coupled with one of the limbs or with a central terminal as is done in precordial electrocardiography.

Normally, as the electrode rises from the lower esophagus, near the diaphragm, the auricular waves are very slight until the electrode reaches a point just behind the left auricle. Then very sharp P waves



appear (Fig. 146). This makes it very simple to know that the electrode is lower than the auricle and therefore adjacent to the posterior portion of the left ventricle. From this latter position the ventricular complex normally should display a prominent R wave, possibly preceded by a

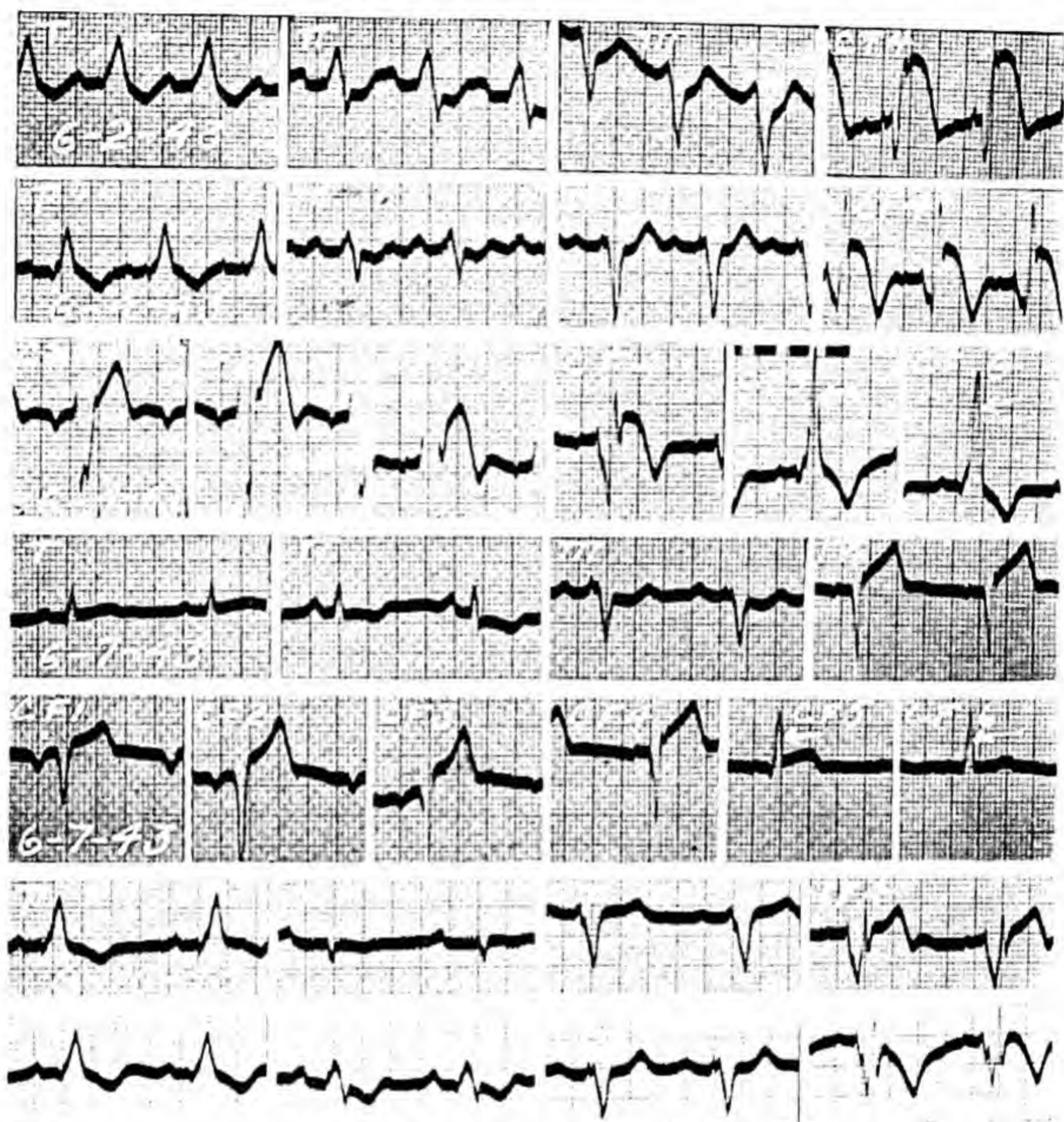


Fig. 145.—Anterior Myocardial Infarction with Transient Left Bundle Branch Block. The first set was made a few hours after the onset of an attack of acute coronary thrombosis. Note the left bundle branch block.  $CF_4$  is most unusual as it shows a very high R-T junction and a prominent  $Q_4$ . Both of these are extremely rare in left bundle branch block and probably indicate infarction of the septum. The six precordial leads on June 4, 1943, show a late intrinsic deflection (arrow) over the left ventricle, confirming diagnosis of left bundle branch block. Curves on June 7, 1943, show that the bundle branch block is gone. Now the intrinsic deflection (arrow) is early. The lowest set shows that finally the  $T_4$  becomes sharply inverted. The patient was a man, seventy-five years of age, who had an operation for hernia under spinal anesthesia. Pre-operative blood pressure was 190 systolic and 110 diastolic, but quickly became imperceptible. One hour after the fall in pressure, as operation was completed, pain in the chest was first felt. The patient then ran a typical course of acute coronary thrombosis and recovered satisfactorily.

very small Q and followed by an upright T. If the posterior wall is infarcted the ventricular complex will show the same changes that are found with an anterior infarction when the precordial electrode is placed over the precordium, *i.e.*, an absent R, an inverted T (Fig. 146), and possibly a deviation of the S-T segment during the acute stages. Esoph-



ageal electrocardiograms obviously cannot be made routinely, as the procedure is somewhat annoying and should not be tried if the patient's condition is too critical. There are occasions, however, when the standard leads are not diagnostic and the clinical condition is such that this procedure would not only be warranted but exceedingly valuable.

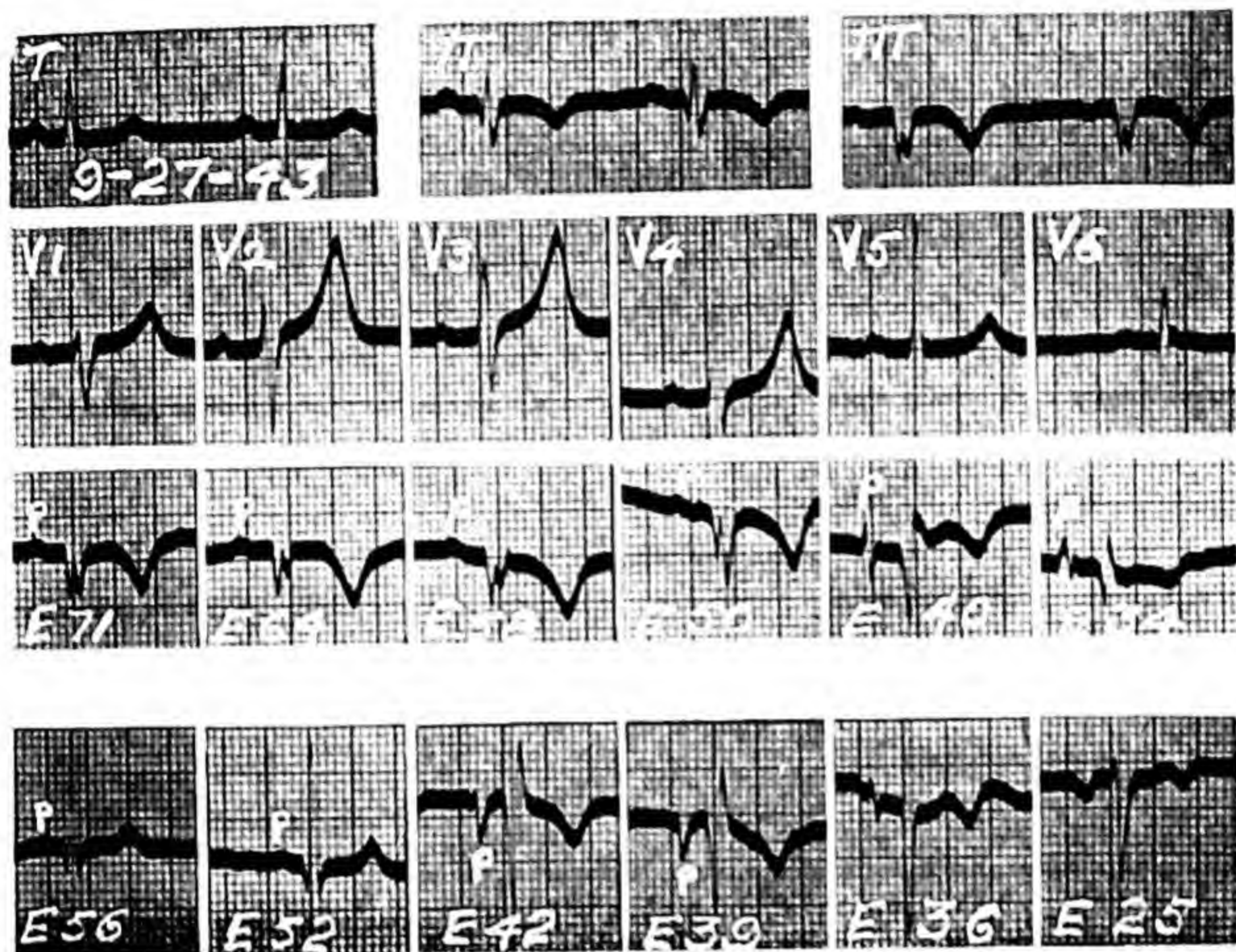


Fig. 146.—Esophageal Leads—Posterior Infarction. The upper set shows three standard leads suggestive of a posterior infarction. The next set shows the six precordial leads using the central terminal, and gives no positive evidence of infarction. The third set was taken with an electrode in the esophagus at various levels from the external nasal orifice (number of centimeters). Note that at distance E 71 to E 50, when the electrode was adjacent to the left ventricle, there were no R waves and T waves were inverted. Contrast these with esophageal curves from a normal individual shown in the lowest set (E 56 and E 52). The appearance of sharp P waves marks the presence of the electrode behind the left auricle and only ventricular complexes below this point are significant. The upper three tracings afford positive proof of posterior infarction.

## PHONOCARDIOGRAPHY

Just as electrical instruments were devised to register the action currents in the heart, similarly apparatus is now available to photograph the heart sounds and murmurs. These instruments are called *phonocardiographs* or *stethocardiographs*. They are coming into use more and more, and it seems appropriate to touch upon the subject of phonocardiography rather briefly.

Heart sounds and murmurs can be augmented in intensity many fold so that they can become audible in an amphitheater or they can be picked up by many listeners by means of appropriate electrical wiring and ear pieces. In fact, heart sounds can be transmitted long distances. These various procedures are used very rarely, but the future may see many



developments little thought of at present. What concerns us here is the photographic registration of the heart sounds as commonly obtained by the simple phonocardiographs in use.

At the outset one might inquire as to what use phonocardiography might have at present. If the physician is hard of hearing the heart sounds can be intensified so that he may be able to detect sounds that he otherwise would not discover. If hearing is normal it is doubtful whether any clinical interpretation of significance can be made from eliciting faint

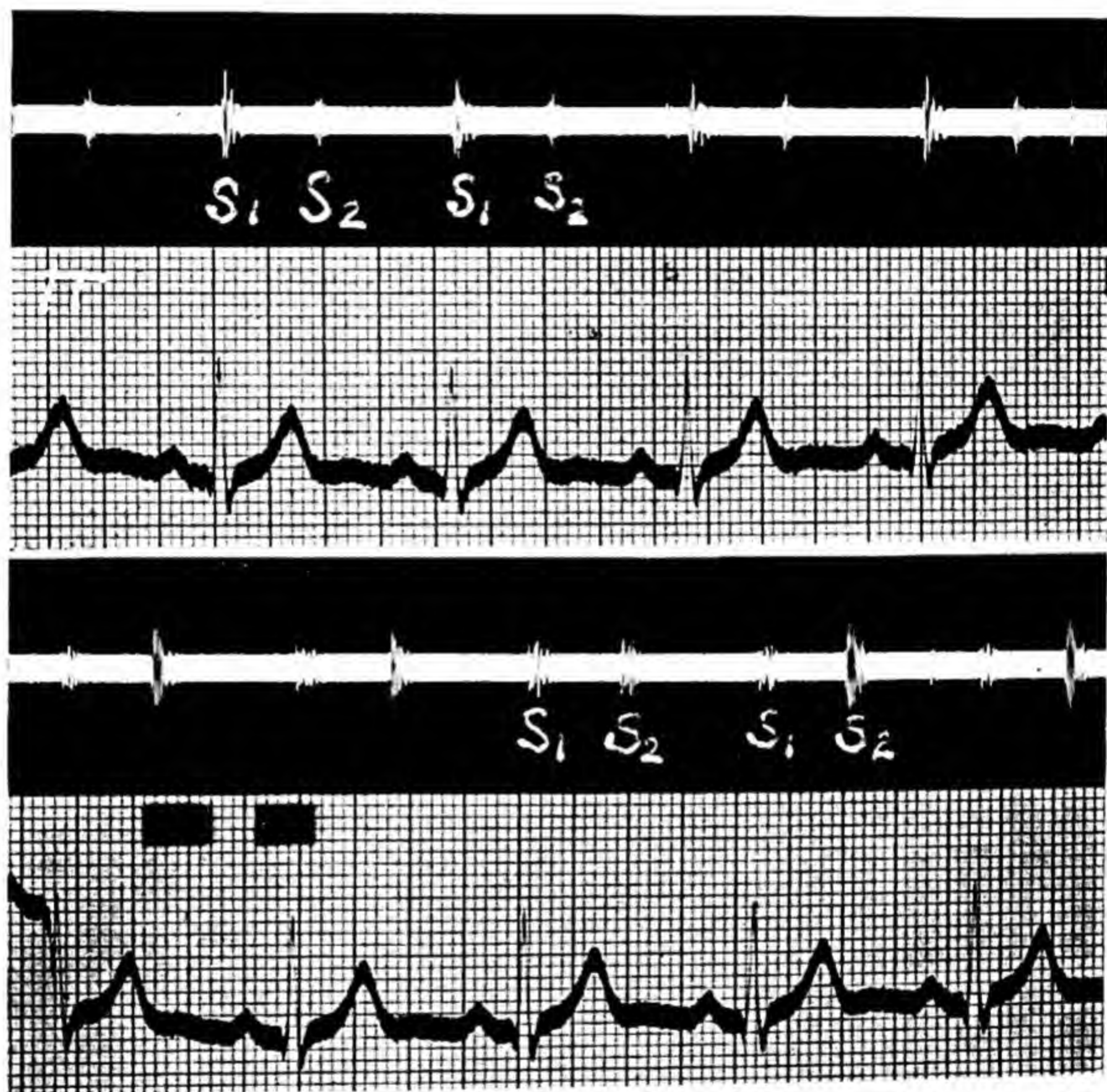


Fig. 147.—Normal Heart Sounds. Simultaneous phonocardiograms and electrocardiograms of a normal man twenty-nine years old. The upper sound curves were taken from the apex and the lower from the pulmonary area. Note that the first sound ( $S_1$ ) is louder at the apex and fainter at the base than the second sound ( $S_2$ ). The relative intensity of the two sounds varies both in normal and abnormal hearts.

sounds with the phonocardiograph that are inaudible with the ordinary stethoscope. There are instances, however, when even the expertly trained ear finds it difficult or even impossible to accurately time a sound or murmur in the cardiac cycle. This is particularly true of the third heart sound producing a gallop rhythm. All physicians have occasionally called a murmur systolic in time erroneously instead of diastolic or vice versa. The simultaneous registration of heart sounds and electrocardiograms obviates any such difficulties, for it enables one to place any of the auscultatory phenomena in their exact portion of the cardiac cycle.



The significance and composition of the heart sounds are being elaborately investigated by many students and will not be discussed in any detail here. Suffice it to say that the first heart sound ("lub") is made primarily by the closure of the mitral and tricuspid valves. It is generally thought that it is partly muscular in origin although some doubt this. The second heart sound ("dub") is made by the closure of the aortic and pulmonary valves. The interval between "lub" and "dub" is systole and that between "dub" and "lub" is diastole. In some normal hearts, especially in young persons in whom the rate is slow, a normal third heart sound may be heard in diastole. The auricular contraction may produce a sound that is rarely audible though it may be present in phonocardiographic tracings. When abnormal sounds or murmurs are heard it is obviously important to place them correctly in one part or another of the cardiac cycle.

Figure 147 is an example of the normal heart sounds obtained in a regular normal heart. It should be noted that the first heart sound comes

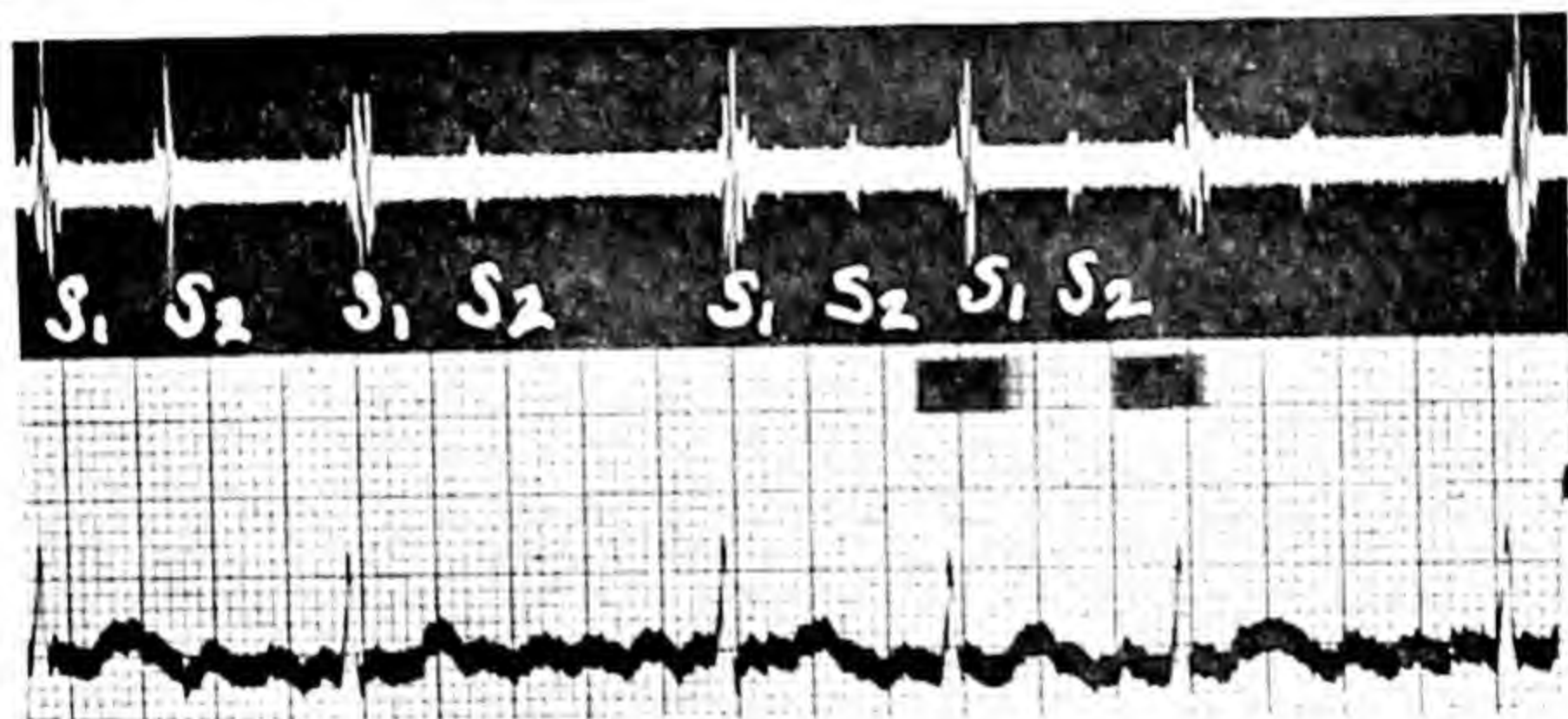


Fig. 148.—Irregular Sounds of Auricular Fibrillation. Note the gross irregularity of the rhythm. Systole ( $S_1$  to  $S_2$ ) is fairly constant but diastole ( $S_2$  to  $S_1$ ) varies from cycle to cycle. The patient was a woman, fifty-six years of age, with thyrotoxicosis.

directly after the QRS complex and the second sound at the very end of the T wave. Figure 148 shows the character of the heart sounds in a grossly irregular heart (auricular fibrillation). The height of vibrations is some indication of the loudness of the sounds. Figure 149 is a clear example of a so-called "normal" mid-systolic gallop rhythm. The extra or abnormal sound occurs in mid-systole, *i.e.*, between the normal first and second heart sound. This must be clearly differentiated from a diastolic gallop, which almost always indicates a pathologic state, in which the third or abnormal sound occurs somewhere between the second and the following first heart sound (Fig. 150).

Pulsus alternans, which has been discussed previously, is detected in the peripheral pulse. Occasionally, however, the alternating quality of the strength of consecutive beats in a regular heart may be detected in the alternate intensity of the heart sounds. This is well displayed in Figure 151. In the upper tracing the alternation in the heart sounds is



accompanied by alternation in the form of the electrocardiogram. There probably is 2:1 defective intraventricular conduction. The lower tracing shows that the auscultatory alternation is still present when the electrocardiograms remain of the same form throughout. This alternation in the loudness of the heart sounds was first detected on routine physical

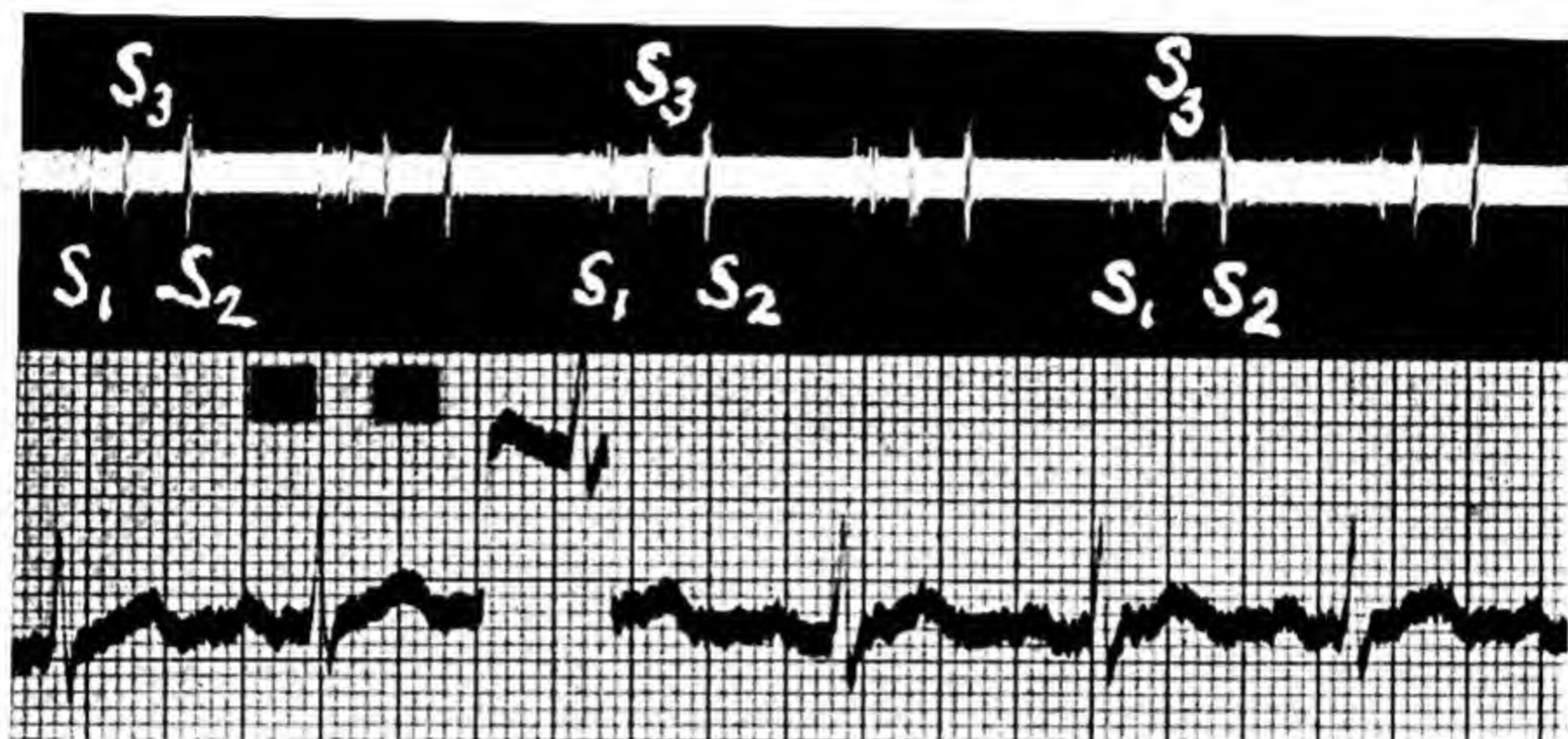


Fig. 149.—Normal Mid-systolic Gallop.—Note that between the normal first heart sound ( $S_1$ ) and the normal second sound ( $S_2$ ) there is a definite third sound ( $S_3$ ), which is even louder than the first sound. The patient was a woman, thirty-five years of age, with rheumatoid arthritis but with no heart disease.

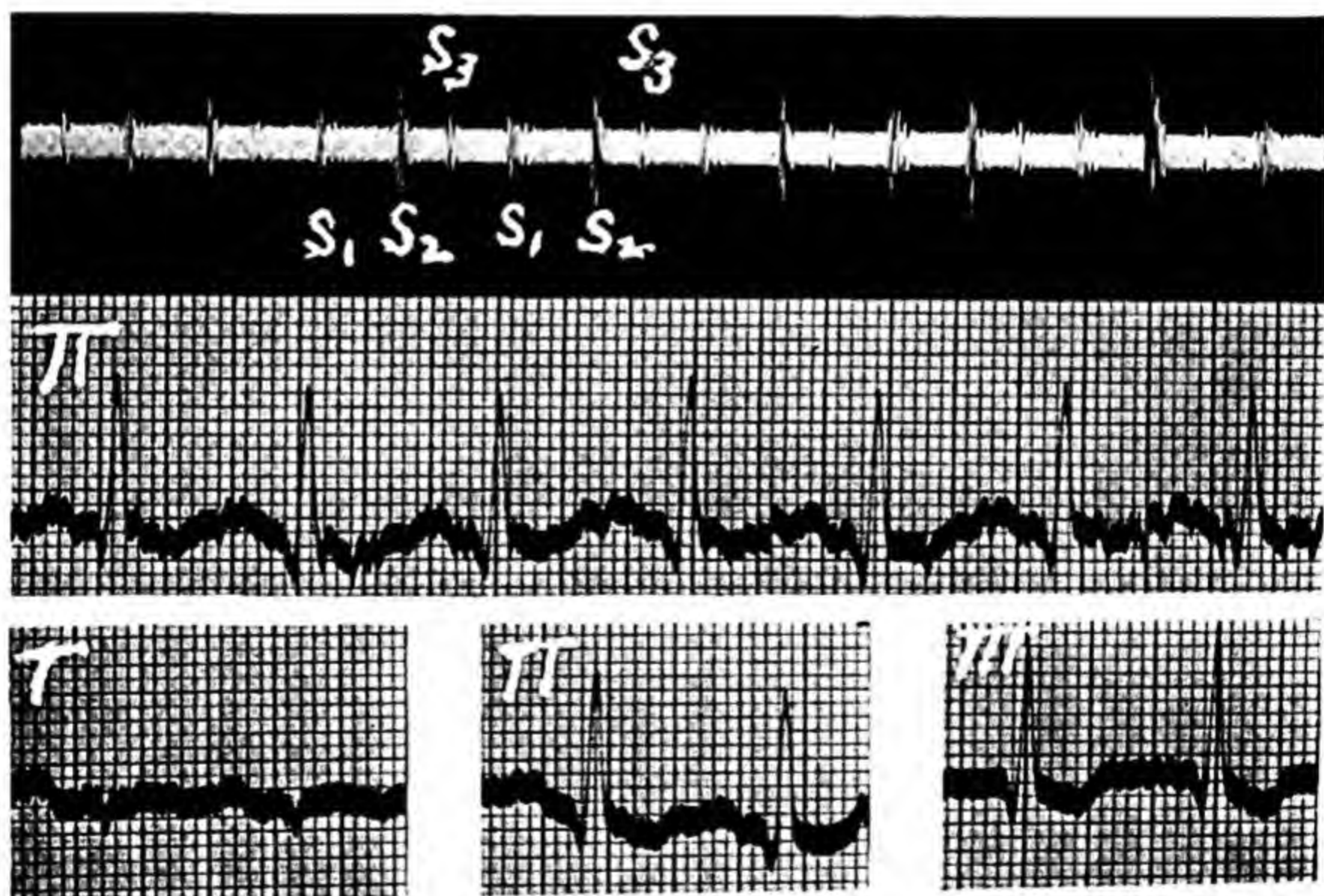


Fig. 150.—Diastolic Gallop Rhythm. Note that the extra or third sound ( $S_3$ ) occurs in diastole, *i.e.*, between the second and first sound. This is almost invariably pathologic. The patient was a man, sixty years old, who had severe congestive heart failure following an acute coronary thrombosis.

examination. Murmurs, if present, may alternate in intensity and the apex impulse may be seen or felt to alternate in strength.

The character and intensity of the first heart sound is a matter of some importance in clinical auscultation. We know it is accentuated in mitral stenosis, hyperthyroidism, and in some nervous and other states. It may



be diminished in intensity in emphysema, pericardial effusion, in acute coronary thrombosis, and, in fact, in some normal, healthy individuals. Amongst the various factors that determine the intensity of the first heart sound, one that is most important is the P-R interval of that beat. It has been shown that when the auriculo-ventricular conduction time varies in different cycles (normally 0.14 to 0.2 second) the first heart sound will be loudest in those cycles with a P-R interval of about 0.04 to 0.08 second and that as the interval becomes longer the sound becomes

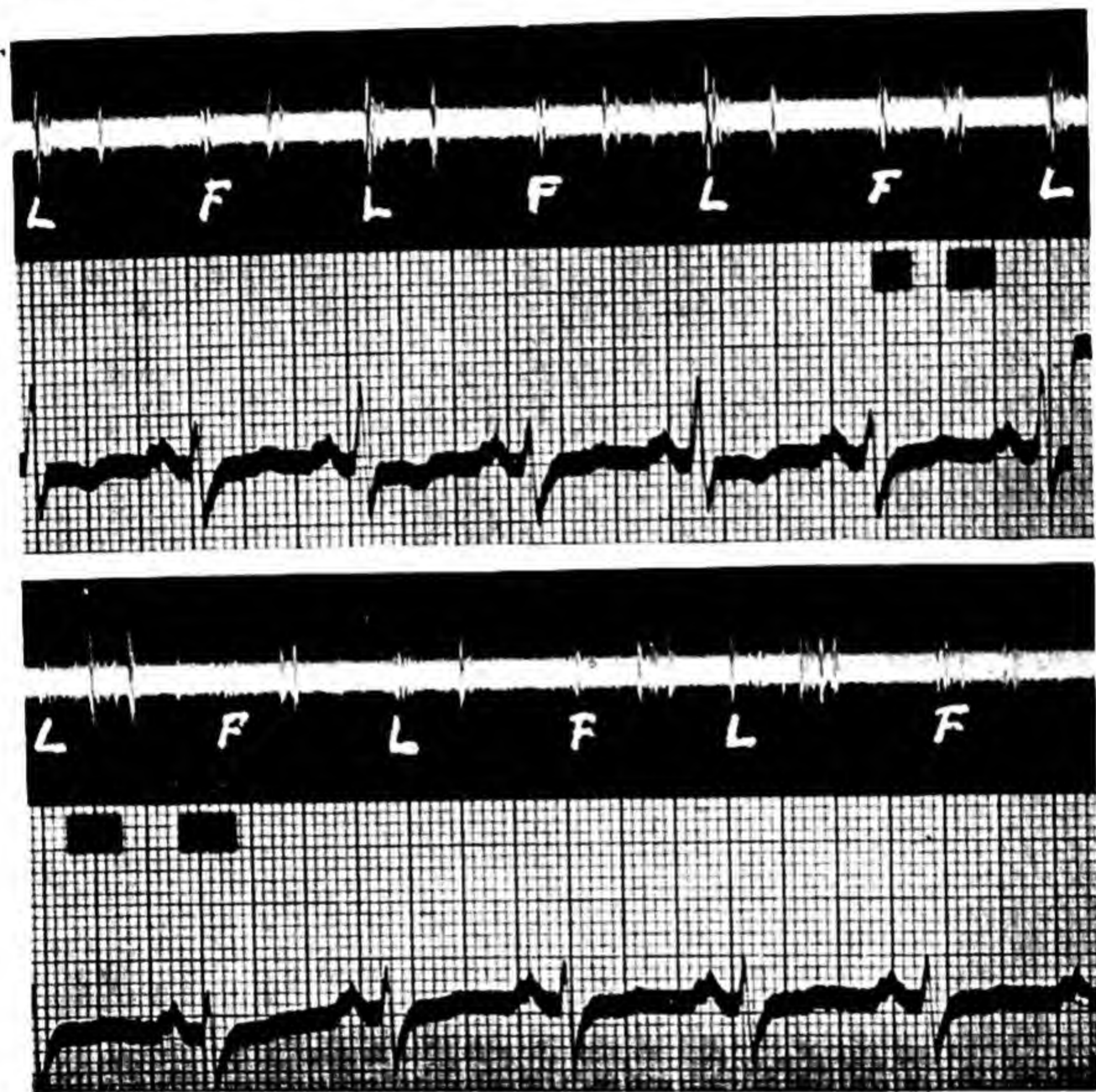


Fig. 151.—Ventricular Alternation. Note that alternate beats have faint (F) and loud (L) sounds. In the upper strip the ventricular complexes also alternate but in the lower set the electrocardiograms are of constant type. The alternation in the heart sounds was audible with the stethoscope and there was also pulsus alternans in the radial artery. This man was seventy-three years old and had serious hypertensive and coronary artery disease.

more distant. It follows that with intervals shorter than normal the sound is louder and with intervals longer than normal the sound becomes fainter than the normal first heart sound.

This method, though not easily applicable, is in fact the only bedside method available in judging abnormalities in the relation between the time of auricular and ventricular contractions. It may aid in the diagnosis of first, second and third degree heart block, nodal premature beats and paroxysmal ventricular tachycardia. There is reason to believe that the changes in the intensity of the first heart sound in these conditions



result because of differences in actual position of the auriculo-ventricular leaflets (mitral and tricuspid) at the moment the ventricles contract. As the ventricles fill in diastole the valve leaflets gradually float upwards and then the auricles contract pushing them deeper into the ventricular cavity, or at least to a different position. The exact position these leaflets will obtain will necessarily be different if the ventricles contract immediately after this alteration resulting from auricular contraction than if a longer interval intervenes between auricular and ventricular contractions. In other words, it seems that the exact position of the mitral and tricuspid valves at the time of ventricular systole determines the loudness of the first heart sound. The character of the snap will be different if it comes from a low position than from a high position. This changing intensity of the first heart sound, while the ventricle is beating quite regularly, is the pathognomonic auscultatory sign of complete heart block and is clearly illustrated in Figure 152. Note that when the P waves

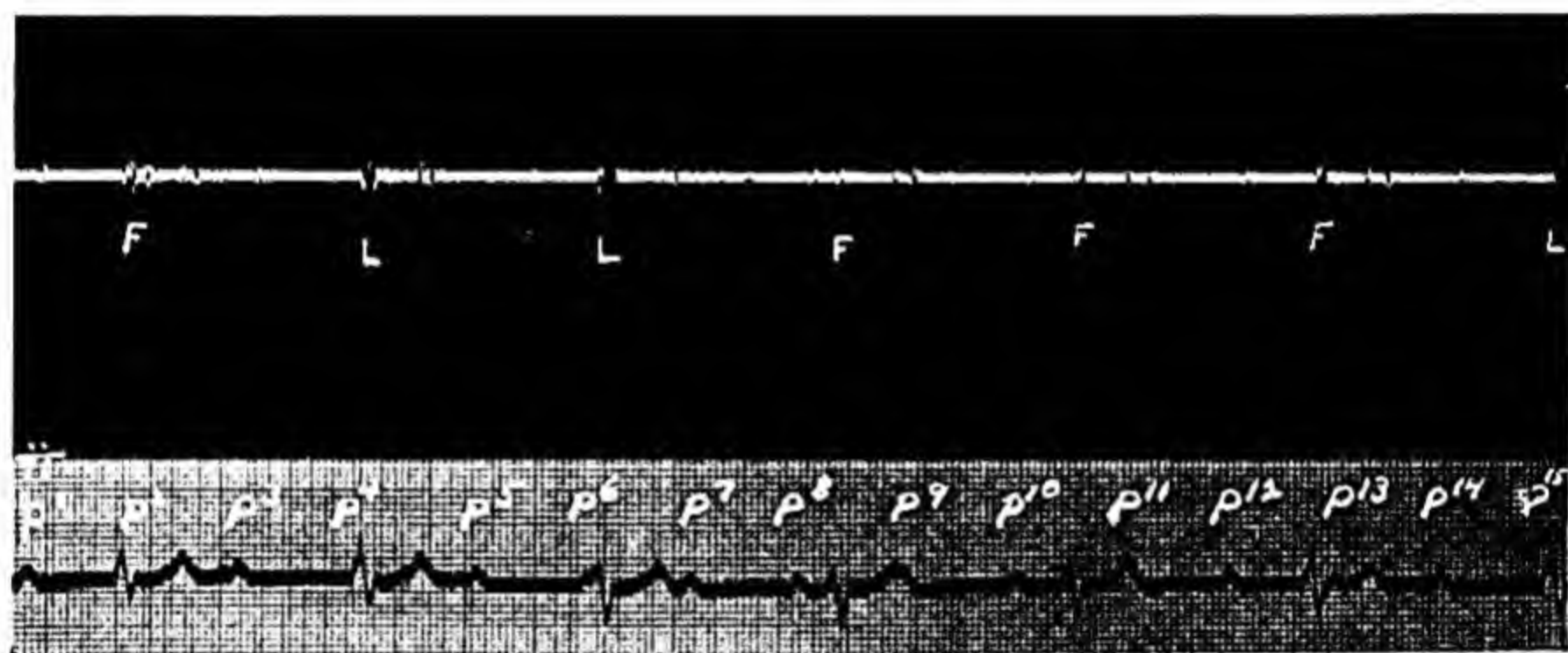


Fig. 152.—Changing First Heart Sound in Complete Heart Block. Note that when the auricular contraction (P) occurs just before the ventricular beats the corresponding heart sound is loud (L) and when the P-R interval is long the heart sound is faint (F). Loud sounds occur at P<sub>4</sub>, P<sub>6</sub> and P<sub>16</sub>. The patient was a woman sixty-nine years old with complete heart block but without heart failure.

come very close to the QRS the first heart sound is very loud and when the P-R interval is long the sound is diminished.

Numerous other illustrations can be given showing the application of phonocardiography to the study of heart sounds. Much of it is still in the speculative stage. The above cases were examples in which the ordinary ear with a simple stethoscope was able to detect significant abnormalities and make correct diagnoses, which were merely confirmed by the phonocardiograph. Possibly much more valuable data will be obtained as such studies continue.

Phonocardiography also affords a simple means of registering cardiac murmurs. With the present methods of amplifying sounds it is an easy matter to transform a faint murmur into a loud roar. That does not help the practising physician, for he will remain dependent for the most part on what he can detect with the ear and a simple stethoscope. He will not be carrying around a complicated machine like the present stethocardi-



graph from house to house, though he may want to use it on occasions for special purposes or for investigative work. Furthermore, the quality of the sounds is not quite the same when electrical apparatus is introduced. Finally, most murmurs that will have any significance or of which any intelligent interpretation can be made will be audible with a stethoscope without electrical amplification. If an extremely faint systolic murmur can only be detected with special apparatus it would have no clinical importance, because even slightly louder systolic murmurs (grade I or I minus) can be heard with the ear and yet often have no pathologic meaning. However, we do know that a diastolic murmur that is diagnostic of mitral stenosis may become so faint that one observer may and another may not hear it, and, in fact, it may not be present at all. Under

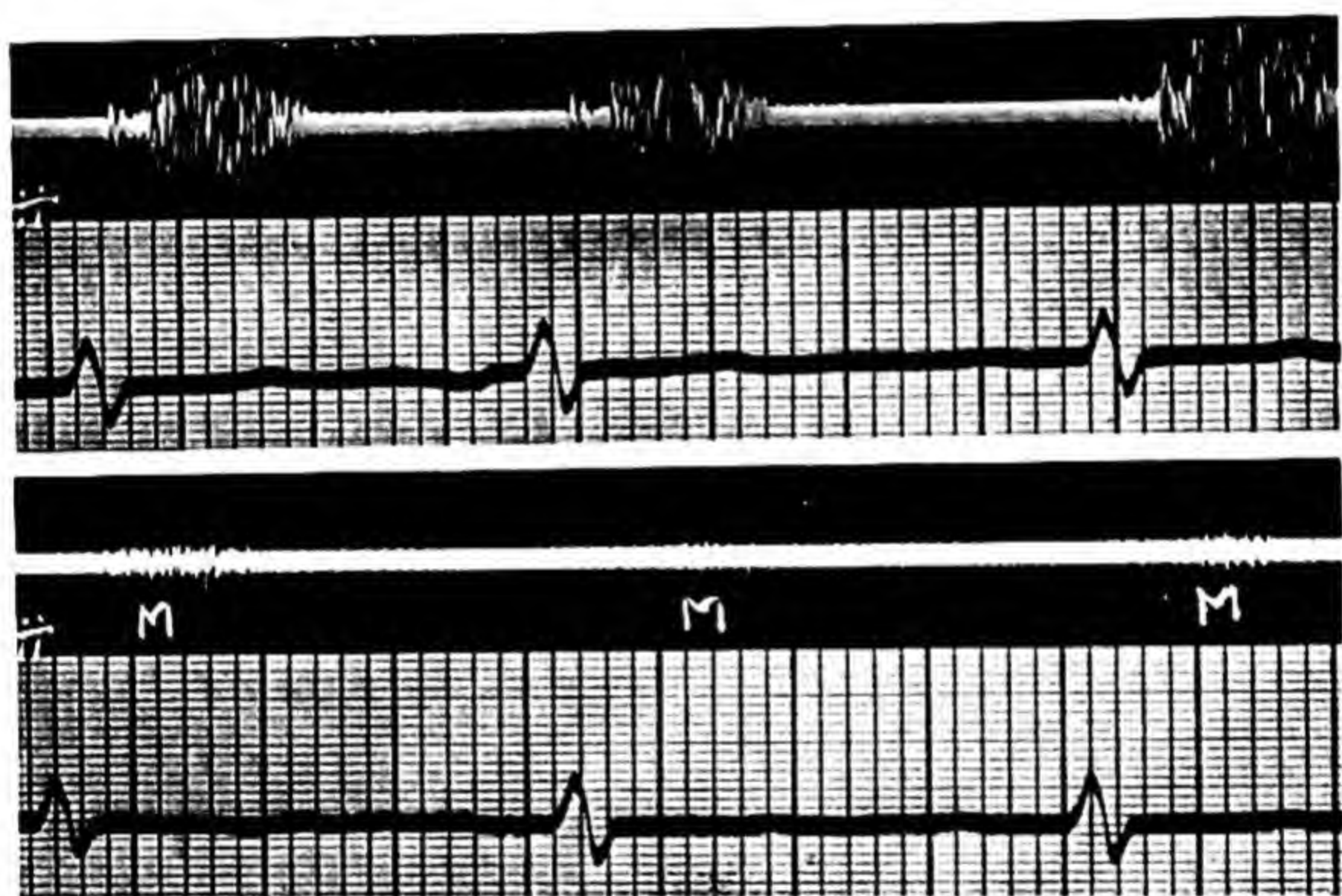


Fig. 153.—Loud Systolic Murmur of Aortic Stenosis Transmitted to the Elbow. The upper tracing shows very loud systolic murmur (grade VI) in the aortic area. The lower sound tracing was taken from the right olecranon process while the blood-pressure cuff was inflated above the systolic pressure. Note that the systolic murmur is still detectable (M). The patient was a woman forty-five years old with aortic stenosis and angina pectoris.

such circumstances amplification and registration of sounds may be valuable clinically. Exploration of such problems is much in need at present.

In a previous discussion (Chapter 17) the significance of systolic murmurs was taken up. At this point it seems appropriate to illustrate graphically one or two points concerning murmurs and to comment about their method of transmission. I have long been convinced that there was something fallacious in the prevailing teaching concerning heart murmurs. It is generally thought that murmurs are transmitted *with* the blood stream. A loud basal systolic murmur if heard in the carotid area is supposed to be indicative of aortic stenosis and an apical systolic murmur if transmitted to the axilla indicative of mitral insufficiency. These diagnoses have been made with greater assurance if such



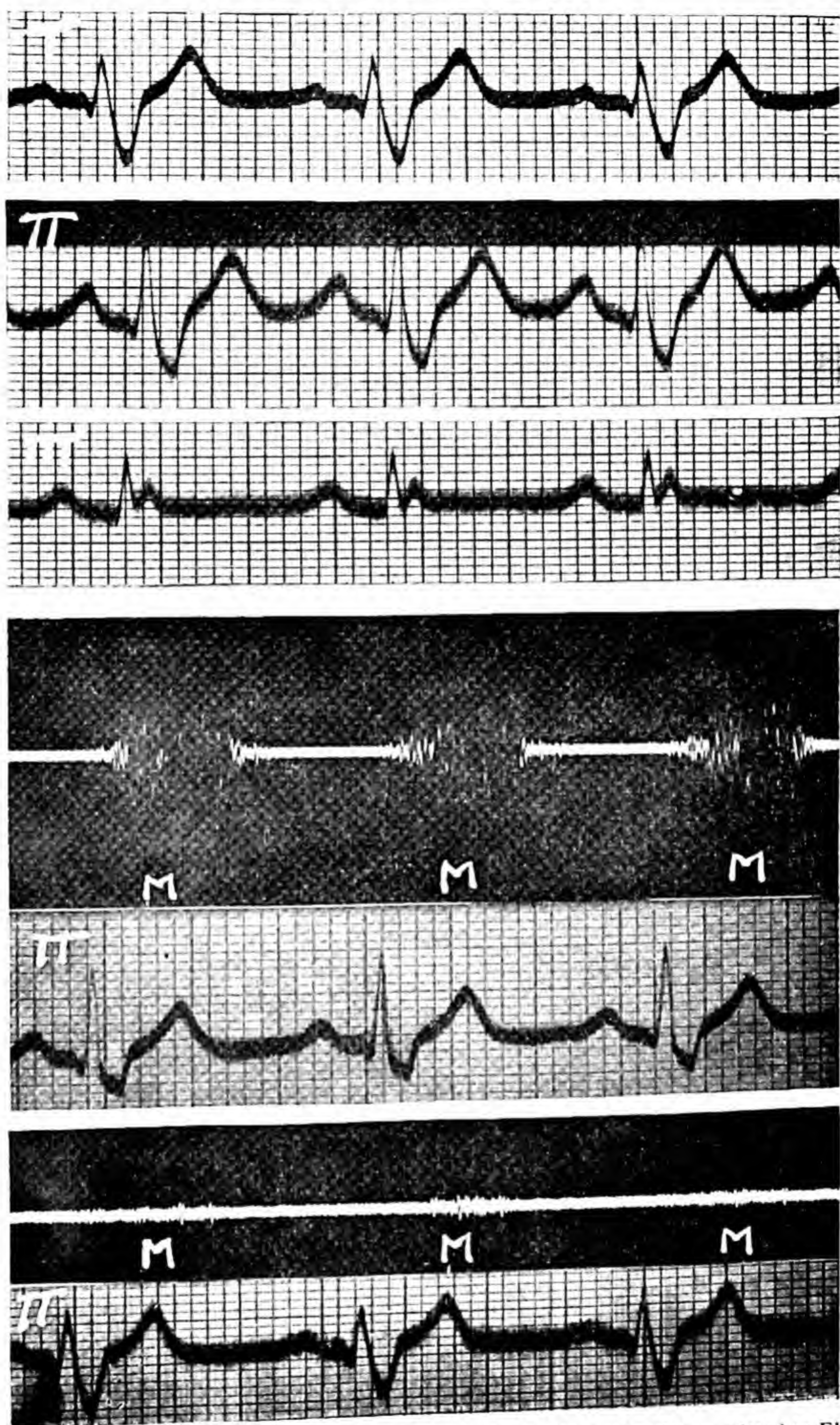


Fig. 154.—Loud Systolic Murmur of Interventricular Septal Defect Transmitted to Elbow. The upper three strips are the three electrocardiographic leads. The middle set shows a loud systolic murmur from the third left sternal border. The lowest set shows that the murmur (M) is audible at the left olecranon process with the blood-pressure cuff inflated above the systolic pressure. The patient was a man, twenty-one years of age, in good health, showing definite evidence of congenital interventricular septal defect.

transmission were present than if not. The point I should like to make is that transmission is dependent mainly, if not entirely, on the loudness of



the murmur, and that transmission takes place from the point of maximal intensity (wherever the origin may be) in all directions. Furthermore, there is evidence now available to show that transmission through bone is most likely of primary importance.

Loud murmurs over the aortic area are often heard in the neck because they are near the neck and loud apical murmurs in the axilla because they are near the axilla. There is no physical reason to explain the transmission of a murmur with the blood stream because the speed of transmission of sound is a great deal faster than the velocity of blood flow. A murmur may be transmitted *in* but not *with* the blood stream. It is not likely even that the transmission in the fluid media is important as will be seen by the following. Figure 153 shows a loud systolic murmur, heard in the aortic area in a patient with marked aortic stenosis.



Fig. 155.—Loud Aortic Diastolic Murmur Transmitted to Elbow. The left set of curves shows loud musical aortic diastolic murmur taken simultaneously with Leads I and II. The right set shows that the diastolic murmur (M) was audible over the right olecranon process with the arterial pressure occluded to that arm. The patient was a man, about fifty years old, who had aortic insufficiency, probably traumatic in origin.

It was also present over the carotid arteries but was also readily heard at the right olecranon process, even when the blood-pressure cuff was inflated to 220 millimeters of mercury (far above the systolic pressure of the patient). This means that the systolic murmur was transmitted through the bones of the arms, for the blood supply to the elbow was entirely cut off.

The fact that the loudness rather than its site of origin determines its transmission is shown by Figure 154. Here a very loud systolic murmur (grade VI) was heard all over the precordium, best in the third left interspace. The patient had congenital interventricular septal defect. This murmur was therefore made within the heart itself, and the current of blood producing the murmur flowed from left ventricle to right ventricle. Despite this the murmur was heard in the carotid artery and also



at the left elbow while the arterial supply was cut off. This murmur also must have been transmitted through bone and not the blood stream. Additional evidence that murmurs are well transmitted through bone is apparent in Figure 155. Here a loud, musical, aortic diastolic murmur was readily audible on top of the head and over the bones of the arms. It is well shown in the phonocardiogram obtained from the right olecranon process.

In general it may be stated that loud murmurs are transmitted in all directions from their maximum point of origin and that bone is probably the main pathway of peripheral conduction. This explains why the systolic murmur of coarctation of the aorta is well heard in the interscapular

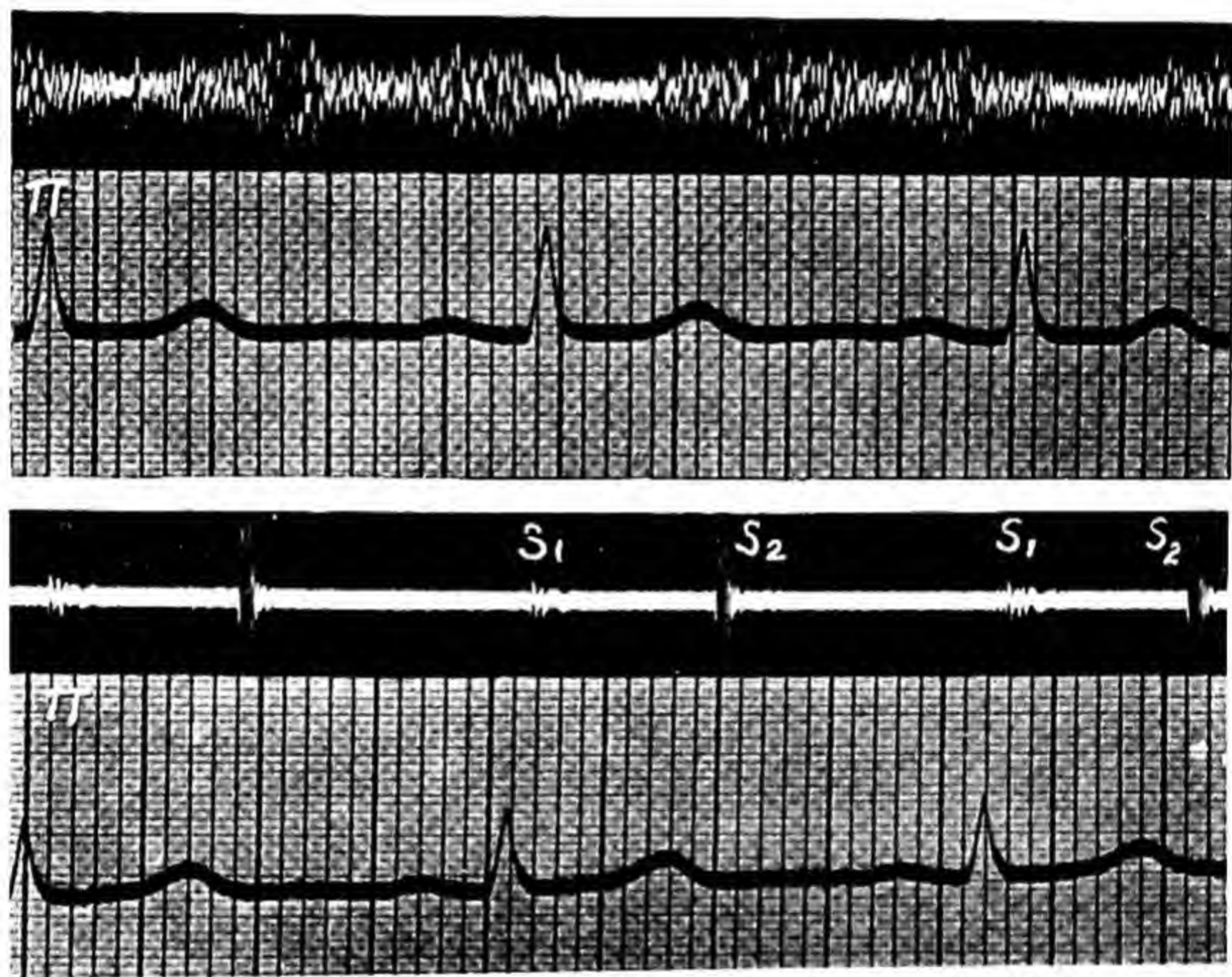


Fig. 156.—Continuous Machinery Murmur of Patent Ductus Arteriosus. The upper set shows a coarse continuous murmur increasing in intensity on approaching the second heart sound and again in the presystole, obtained from the pulmonary area. The lower tracings show normal first and second sounds ( $S_1$  and  $S_2$ ) without any murmurs following successful surgical division of the ductus. The patient was a woman, twenty-four years of age, with congenital patent ductus botalli, who is now cured.

region, for the site of formation of the murmur is deep in the chest and near the spine. It follows that any murmur that is loud enough, no matter what its origin, may be audible over the carotid artery or even over the bones of the arms.

Apart from the intensity of murmurs one derives very little diagnostic value from the quality of murmurs. At times, however, the peculiar quality is of some interest. This is particularly true of the continuous machinery murmur of patent ductus arteriosus. This murmur often seems to envelop the heart sounds, becoming a little louder as the second heart sound approaches and continuing uninterruptedly after the second sound. In this way it differs from the systolic and diastolic mur-



murs of aortic stenosis and insufficiency, in which there appears to be two separate components. Figure 156 shows the disappearance of such a continuous murmur in a case of patent ductus arteriosus after a successful surgical division of the duct.

One or two more simple observations may be appropriate at this time. It has been mentioned in Chapter 17 that slight systolic murmurs may result from anemia, fever, hyperthyroidism, physical exercise, emotion and other non-cardiac causes. Figure 157 is a simple example of the production of a systolic murmur by fever. This patient had no evidence of heart disease and showed no murmurs whatever. She had general paresis and was receiving malaria therapy. During the height of the fever

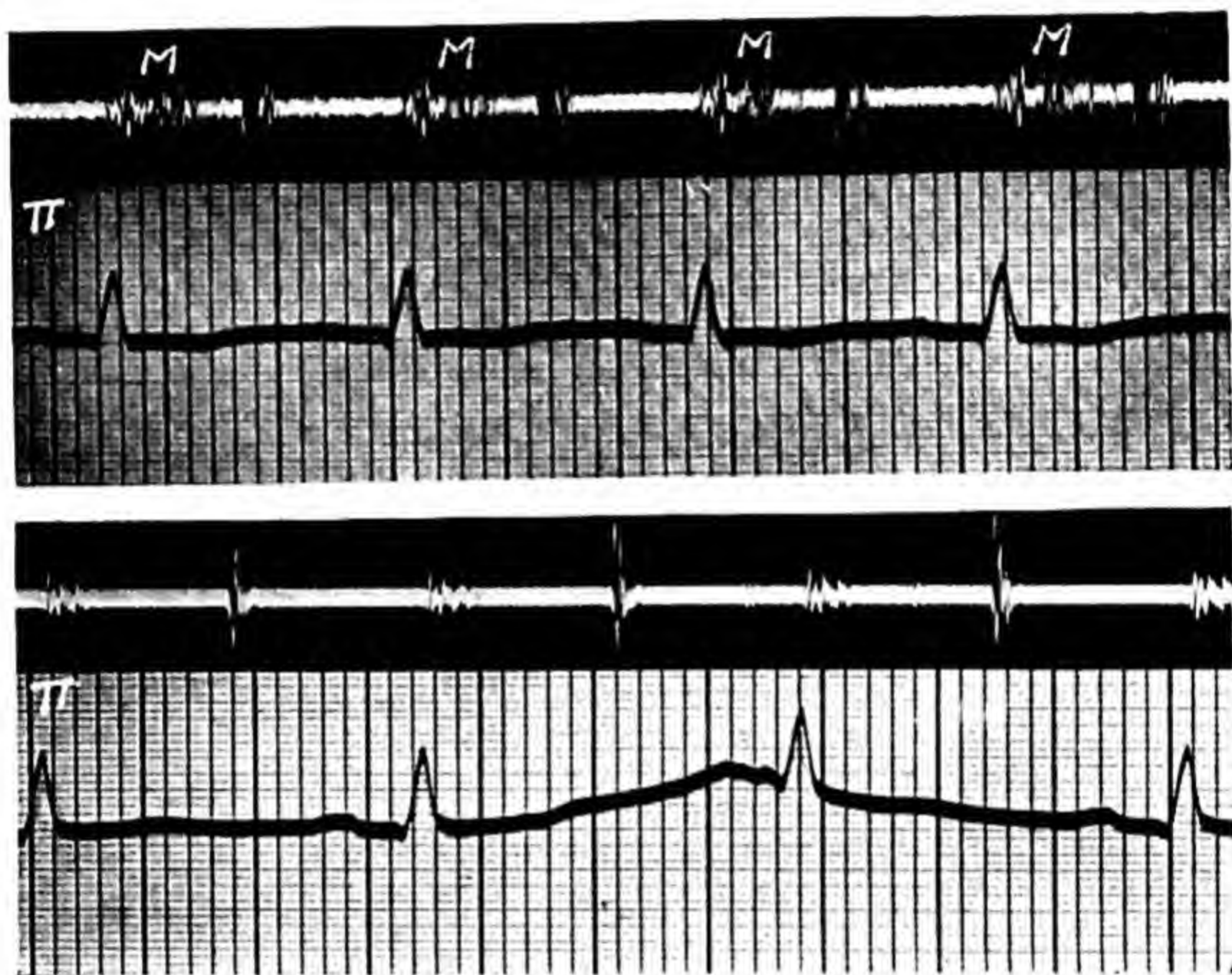


Fig. 157.—Systolic Murmur Produced by Fever. The upper set shows systolic murmur (M) in the pulmonary area while the patient's temperature was 104° F. The lower set shows no murmur when the patient's temperature was 99.2° F. This patient had general paresis but no heart disease and was receiving malaria therapy.

(temperature 104° F.) a distinct basal systolic murmur was present that entirely disappeared as the temperature returned to normal. Finally, it is well to bear in mind that the disappearance of a murmur with inspiration does not necessarily mean that it has no clinical significance. Many faint systolic murmurs will vary in intensity with respiration and some will disappear with a deep inspiration. However, a faint diastolic murmur that is diagnostic of organic valvular disease may also disappear after a deep breath. The procedure obviously decreases the intensity of some murmurs and the very faint ones may therefore become inaudible.

Much more could be discussed concerning murmurs and their registration by phonocardiography but the entire subject is in the process of investigation and awaits further elucidation.



## INDEX

- ABDOMINAL belt, in angina pectoris, 101  
     findings, in coronary thrombosis, 110, 111  
     pain in pericarditis, rheumatic, 57  
     and tenderness, in rheumatic fever, 11  
     paracentesis, for congestive heart failure, 282  
     symptoms, in rheumatic fever, 28  
 Aberrant renal artery, hypertension and, 136  
 Abortion, indication in cardinals for, 239-244  
 Accidents, coronary disease and, 215  
     heart disease and, 214-217  
 Acetyl- $\beta$ -methylcholine, for paroxysmal auricular tachycardia, 193  
 Acid ash diet, for congestive heart failure, 270  
 Adams-Stokes disease, adrenalin in, (Fig. 62) 352, 353  
     in complete heart block, 364, 365  
     in coronary thrombosis, 121  
     "status epilepticus" in, 206  
     syncope in, 206  
     treatment of, 206, 207  
     ventricular fibrillation in, (Fig. 62), 352  
     "flutter" in, (Fig. 60), 350  
 Addison's disease, cardiac features of, 141  
 Adhesive pericarditis, 72-76. See *Pericarditis, chronic non-constrictive*.  
 Adrenal tumors, hypertension and, 127, 136  
 Adrenalin, in Adams-Stokes disease, (Fig. 62) 206, 207, 352, 353, 366, 367  
     angina pectoris and, 84  
     in coronary thrombosis, 120, 121  
     test, for angina pectoris, 91  
     ventricular fibrillation and, 351  
 Age, at death, in aortic stenosis, 55  
     in mitral stenosis, 55  
     in tricuspid stenosis, 55  
 Albuminuria, in lupus erythematosus disseminatus, 141  
 Alcohol, in angina pectoris, 96  
     injections of dorsal roots, in angina pectoris, 102  
 Allergic state, heart disease and, 219, 220  
     in rheumatic fever, 7  
 Alpha-lobeline, for velocity of blood flow determination, 258  
 Alternation of apex impulse, 132  
     in pulsus alternans, 413  
     of auricular complex, 414  
     of heart sounds, 133  
     in pulsus alternans, (Fig. 151) 413, 429  
     of ventricular complex (electrical alternans), (Fig. 151) 413, 414, 431  
 Aminophyllin, in angina pectoris, 101  
     for Cheyne-Stokes breathing, 262  
     for congestive heart failure, 277  
     in coronary thrombosis, 122  
     intravenous, for paroxysmal dyspnea, 135  
 Ammonium chloride (enteric coated), for congestive heart failure, 278, 279  
 Amyl nitrite, in angina pectoris, 96  
     test, for presystolic murmur, 32  
 Anemia, angina pectoris and, 77, 81  
     dilatation of heart and, 29, 223  
     dyspnea caused by, 223  
     in rheumatic fever, 14  
     systolic murmur in, 229  
     velocity of blood flow and, 146  
 Anesthesia, paroxysmal auricular tachycardia during, 192  
     peripheral circulatory failure in, 252  
     for thyrotoxic heart disease, 148  
 Anesthetics, in surgical operations, 238  
 Aneurysm of aorta, angina pectoris confused with, 91  
     dyspnea in, 225  
     velocity of blood flow in, 259  
     of ventricle, following coronary thrombosis 113  
 Angina pectoris, 76-105  
     adrenalin and, 84  
     test for, 91  
     anemia and, 77, 81  
     aneurysm confused with, 91  
     anoxemia of myocardium as cause of, 77, 90  
     test for, 90  
     aortic insufficiency and, 81  
     stenosis and, 48-50, 81  
     valvular disease and, 78, 103  
     arthritis of spine confused with, 91  
     auricular fibrillation and, 87, 88  
     blood pressure in, 87  
     Buerger's disease and, 78, 81  
     calcification of coronary arteries in, 87  
     cardiac findings in, 88  
     cervical rib confused with, 91  
     cholesterol diet and, 80, 81  
     clinical entity of, 76  
     congestive heart failure and, 94  
     constitutional type in, 11, 12, 78  
     coronary sclerosis and, 83, 87, 106, 107  
     diabetes and, 80  
     diagnosis, differential, 91  
     diagnostic tests for, 90, 91  
     diaphragmatic hernia confused with, 91



- Angina pectoris, electrocardiogram in, 89, 90
- etiological factors, 77-84
  - exercise test for, 90
  - findings during attack of, 86
  - functional heart disease confused with, 91
  - gallbladder disease confused with, 91
  - gout and, 81
  - heart size in, 87
  - heredity and, 77, 78
  - herpes zoster confused with, 91
  - hypercholesteremia and, 78, 80
  - hypoglycemia and, 86, 98
  - Jews and, 79
  - lead poisoning and, 81
  - left hand disability in, 91
    - shoulder pain in, 91
  - mechanism of attacks in, 83
  - myxedema heart and, 81, 137
  - nitroglycerin workers and, 83
  - Paget's disease and, 81
  - paroxysmal rapid heart action and, 81-83
  - peptic ulcer and, 99
  - polycythemia and, 81
  - prognosis in, 92-95
    - age of onset and, 94
    - blood pressure and, 94
    - duration of life, 93
    - electrocardiogram and, 94, 95
    - foci of infection, 94
    - hereditary factor, 94
    - mode of death, 95
    - obesity and, 94
  - rhythm of heart and, 87
  - sex and, 77, 79
  - sexual intercourse and, 85
  - spasm of coronary arteries in, 83
  - subdeltoid bursitis confused with, 91
  - sudden death in, 92, 93, 95
  - symptoms of, 84-87
  - syphilis and, 79, 80
  - syphilitic aortic insufficiency and, 80
    - aortitis and, 80, 154
  - thoracic tumor confused with, 91
  - thyrotoxicosis and, 77, 81, 82, 85
  - tobacco and, 79, 99
  - trauma to chest and, 86
  - treatment of, 95-105
    - abdominal belt, 101
    - alcohol, 96
      - injections of dorsal roots, 102
    - aminophyllin (theophyllin ethylene-diamine), 101
    - amyl nitrite, 96
    - atropine, 100
    - bromides, 100
    - carotid sinus pressure, 96
    - cervical sympathectomy, 102, 103
    - climate, 100
    - cobra venom, 101
    - coronary sinus ligation, 104
- Angina pectoris, treatment of, diet, 98
- digitalis, 100
  - diuretin (theobromine sodium salicylate), 101
  - dorsal sympathectomy, 102
  - erythrol tetranitrate, 101
  - loss of weight, 98
  - morphine, 96
  - nitroglycerin, 95, 96
  - omentoplexy, 104
  - pectoral muscle implantation to pericardium, 104
  - phenobarbital, 100
  - phyllicin, 101
  - potassium iodide, 99, 100
  - quinidine, 101
  - rest in bed, 97, 98
  - sodium amytal, 100
  - surgical procedures, 102
  - testosterone, 101
  - theobrominal, 101
  - theocalcin (theobromine calcium salicylate), 101
  - theominal, 101
  - thesodate (theobromine sodium acetate), 101
  - tissue extracts, 101
  - tobacco, 99
  - total thyroidectomy, 104
  - x-ray of adrenals, 101
  - "trigger" mechanism in, 84
  - ventricular fibrillation and, (Fig. 63) 353
  - vital capacity in, 89
  - xanthomatosis and, 80
- Annulus fibrosus of mitral valve, calcification of, 39
- Anoxemia of myocardium, as cause of angina pectoris, 77, 90
- test, for angina pectoris, 90
  - of tissues, congestive heart failure and, 256
- Antipyretics, for congestive heart failure, 280
- Antistreptolysins in rheumatic fever, 15
- Aortic diastolic murmur, in rheumatic carditis, 30
- insufficiency, 44-47
    - angina pectoris and, 81
    - apex impulse in, 44
    - Austin Flint murmur in, 45, 46
    - capillary pulse in, 46
    - Corrigan pulse in, 46
    - diastolic murmur in, 45
      - thrill in, 44
    - Duroziez's sign in, 46
    - hypertension and, 127, 134
    - pericarditis, rheumatic, confused with 58
    - peripheral signs of, 46
    - "Pistol shot" in, 46
    - pulse pressure in, 46
    - relative, 44, 45
    - retinal pulsations, in, 46



- Aortic insufficiency, rupture of aorta and, 118  
     systolic murmur in, 44, 45  
     ventricular (left) dilatation in, 44  
     "roughening," systolic murmur and, 233  
     second sound, in aortic stenosis, 51  
         in syphilitic aortitis, 152, 153  
     stenosis, 47-53  
         age at death in, 55  
         angina pectoris and, 48-50, 81  
         aortic second sound in, 51  
         basal metabolism increase in, 147  
         blood pressure in, 53  
         calcification of valve in, 51  
         causes of, 47, 48  
         diagnosis of, 50  
         diastolic murmur in, 52  
         duration of symptoms in, 55  
         electrocardiogram in, 53  
         general features of, 48  
         plateau pulse in, 53  
         sinus pauses in, (Fig. 11) 305  
         slow heart in, 50  
         sudden death in, 49  
         syncope in, (Fig. 11) 50, 205, 305  
         systolic murmur in, 51, 52  
         thrill in, 50, 51  
         x-ray in, 53  
     valvular disease, 43-53  
         angina pectoris and, 78, 103  
         auricular fibrillation in, 43  
         conduction disturbances in, 43  
         general features of, 43
- Apex impulse, in aortic insufficiency, 44  
     in coronary thrombosis, 110
- Aphonia, in mitral stenosis, 38  
     in syphilitic aneurysm of aorta, 153
- Appendicitis, acute, rheumatic fever confused with, (Fig. 69) 11, 28, 29, 234, 235, 357
- Arborization block, 373
- Argyria, cyanosis confused with, 171, 172
- Arrhythmias, in coronary thrombosis, 110  
     functional heart disease with, 181, 182
- Arteriosclerosis, gray hair and, 144  
     hypertension and, 129
- "Arteriosclerotic heart disease," 129, 130
- Arteriovenous fistula, 138  
     continuous murmur in, 138  
     pulse pressure elevation in, 138  
     surgery for heart failure due to, 293, 294
- Arthritis of spine, angina pectoris confused with, 91  
     coronary thrombosis confused with, 118
- Ascites, in congestive heart failure, 263  
     in pericarditis, constrictive, 69  
     in tricuspid stenosis, 54
- Aschoff nodule, 7
- Asthma, early graying of hair in, 144
- Atelectasis of lung, in syphilitic aneurysm of aorta, 153
- Atrial (auricular) septal defect, 175
- Atropine, for Adams-Stokes disease, 207  
     in angina pectoris, 100  
     for carotid sinus sensitivity, 208  
     effect on heart block, (Fig. 75) 362  
     in paroxysmal ventricular tachycardia, 121, 349  
     for premature ventricular beats, 345  
     for syncope, 205
- Auricular (left) dilatation, in mitral stenosis, 39
- Auricular (right) dilatation, in tricuspid stenosis, 55
- Auricular fibrillation, angina pectoris and, 87, 88  
     in aortic valvular disease, 43  
     bedside recognition of, 34, 334, 335  
     in beri-beri heart, 139  
     carotid sinus pressure in, (Fig. 35) 326  
     in coronary thrombosis, 110  
     digitalis as cause of, 332  
         effect on, (Fig. 36) 328  
     effect on murmurs of mitral stenosis of, 36  
     electrocardiogram in, 35  
     heart sounds in, (Fig. 148) 429  
     mechanism of, 327  
     in mitral stenosis, 33  
     multiple extra systoles distinguished from, 34, 35  
     nature and effect of, 34  
     paroxysmal, (Fig. 39) 198-201, 330, 331. See also *Paroxysmal auricular fibrillation*.  
     polygram in, 35  
     pregnancy and, 240  
     prevention of, quinidine in, 290  
     pulse deficit in, 34  
     quinidine in, (Figs. 37, 40, 41) 289, 290, 329, 331, 335  
     in scleroderma heart, 140  
     "surgical abdomen," confused with, 335  
     operations in, 237  
     in thyrotoxic heart disease, 143, 145  
     in tricuspid stenosis, 55  
     in tumors of heart, 140  
     without heart disease, (Fig. 37) 183, 329  
         quinidine in, 288
- flutter, 196-198. See also *Paroxysmal auricular flutter*.  
     without block, (Fig. 33) 322, 324  
     carotid sinus pressure in, (Figs. 30, 35) 322, 326  
     in coronary thrombosis, 110  
     digitalis effect on, (Figs. 29, 31, 32, 34) 321-327  
     quinidine, (Fig. 29) 321, 327  
     standstill, digitalis and, (Fig. 14) 306, 307  
         quinidine and, (Fig. 15) 307  
     thrombi, pulmonary emboli and, 211
- Auriculo-ventricular node, 297



- Austin Flint murmur, in aortic insufficiency, 45, 46  
     in syphilitic aortic insufficiency, 154  
 Avertin, in treatment of chorea, 24  
 Avitaminosis, thyrotoxicosis and, 144  
 Axis deviation, clinical interpretation of, 376-378  
     (left), effect of deep inspiration on, (Fig. 96) 377  
     without heart disease, (Fig. 93) 375  
     (right) in mitral stenosis, (Fig. 94) 376  
     in pulmonary stenosis, (Fig. 95) 376  
 Ayerza's disease, 223
- "BACKWARD FAILURE" theory, congestive heart failure, 254
- Bacterial endocarditis, 156  
     bicuspid valves and, 179  
     chronic valvular disease following, 159  
     congenital heart disease and, 173  
     in patent ductus arteriosus, 178  
     post partum, 161  
     ruptured valves of heart in, 138
- Bacterial endocarditis, acute, 157-159  
     normal valves affected by, 158  
     penicillin for, 158, 159  
     sulfa drugs for, 158
- subacute, 159-170  
     age incidence of, 160  
     bacteria free type of, 163  
     bicuspid aortic valves and, 160  
     blood cultures in, 161, 162  
     chronic nephritis from 163  
     clinical features of, 160  
     clubbing of fingers in, 161, 162  
     complications of, 162, 163  
     congenital heart disease and, 159, 160  
     coronary embolism in, 163  
     dicoumarin for, 169  
     differential diagnosis of, 164  
     emboli in, 162  
     extraction of teeth and, 161  
     fever in, 161  
     therapy in, 167, 168  
     "grippe"-like onset of, 161  
     heart failure in, 163  
     rate in, 165  
     hematuria in, 161, 162  
     heparin for, 169  
     neosalvarsan in treatment of, 167  
     onset of, 161  
     painful finger tips in, 161, 162  
     patent ductus and, 160, 163  
     ventricular septum and, 176  
     penicillin for, 168, 169  
     petechiae in, 161  
     postoperative, 161  
     predisposing causes, 159, 160  
     prevention of, 169, 170  
     prognosis of, 166
- Bacterial endocarditis, subacute, pulmonary emboli in, 163  
     rheumatic heart disease and, 159, 160  
     state in relation to, 165, 166  
     rupture of valve in, 163  
     of ventricle in, 163  
     of ventricular septum in, 163  
     scarlet fever and, 160  
     sex incidence of, 160  
     "simple cold" and, 161  
     sore throat and, 161  
     splenic enlargement in, 161  
     splinter hemorrhages in, 162  
     streptococcus skin test in, 165  
     viridans and, 160  
     sulfa drugs for, 167, 168  
     syphilitic aortic insufficiency and, 160  
     symptoms of, 161  
     transfusion for, 167  
     treatment of, 167-69  
     tuberculosis confused with, 164  
     typhoid vaccine in treatment of, 168  
     valves involved in, 163, 164  
     ventricular septal defect and, 160, 163
- Barium chloride, for Adams-Stokes disease, 207
- Basal metabolism, increase of, in aortic stenosis, 147  
     in cardiac failure, 147  
     in fever, 147  
     in hypertension, 147  
     in leukemia, 147  
     thyrotoxic heart disease and, 146, 147
- Beri-beri heart, 138, 139  
     auricular fibrillation, transient, in, 139  
     chronic alcoholism and, 139  
     congestive failure and, 138-139  
     dilatation of heart in, 139  
     electrocardiogram in, 139  
     gallop rhythm in, 139  
     hyperthyroidism and, 139  
     mural thrombosis and, 139  
     pregnancy and, 139  
     pulse pressure increase in, 139  
     vitamin B deficiency and, 139
- Bicuspid valves, bacterial endocarditis and 160, 179  
     in congenital heart disease, 179
- Bifid apex impulse, 132
- Bigeminy, digitalis causing, (Figs. 52, 53) 342, 343  
     premature ventricular beats and, (Fig. 51) 340, 341
- Bleeding. See *Phlebotomy*
- Blood cultures, in bacterial endocarditis, subacute, 161, 162
- Blood pressure, in angina pectoris, 87  
     in aortic stenosis, 53  
     cerebral hemorrhage and, 208, 209  
     in coarctation of the aorta, 174  
     determination of 130



- Blood pressure, mitral stenosis and, 41, 42  
 in patent ductus arteriosus, 177  
 pulsus alternans and, 413  
 in thyrotoxic heart disease, 146  
 in tricuspid stenosis, 55
- Blood pressure fall, cerebral failure from, 106  
 in coronary thrombosis, 109  
 coronary thrombosis from, 106
- Blood volume, in congestive heart failure, 256, 257
- Brachigram, pulse deficit shown by, (Fig. 52) 342
- Bradycardia, after infections, 303  
 in jaundice, 303  
 in normal individuals, 303  
 in sleep, 303  
 in undernutrition, 303
- Brain tumor, cerebral hemorrhage confused with, 208
- Brassy cough, in syphilitic aneurysm of aorta, 153
- Brauer operation, for chronic adhesive pericarditis, 75
- Breathlessness See *Dyspnea*.
- Broadbent's sign, of adhesive pericarditis, 74
- Bromides, in angina pectoris, 100
- Bronchial compression, in mitral stenosis, 38, 39  
 disease, dyspnea in, 219-223  
 cardiac dyspnea confused with, 220-223  
 factor, in dyspnea, 219-223  
 veins, in mitral stenosis, 39
- Bronchiectasis, dyspnea in, 225
- Bronchiogenic carcinoma, confused with mitral stenosis, 38
- Buerger's disease, angina pectoris and, 78, 81  
 cerebral hemorrhage confused with, 208  
 tobacco and, 99
- Bundle branch block, clinical diagnosis of, 370, 373  
 incomplete, 373  
 left, (Figs. 83-86) 368-370  
 left (2:1), (Fig. 84) 370  
 right, (Figs. 87-90)  
 prognosis in, 373
- Burns, peripheral circulatory failure in, 252
- CACHEXIA**, in congestive heart failure, 264
- Caffeine sodium benzoate, 277  
 for cerebral accidents, 210  
 for Cheyne-Stokes breathing, 262  
 in coronary thrombosis, 120  
 for peripheral circulatory failure, 253
- Calcific aortic stenosis, x-ray visualization of, 151
- Calcification of annulus fibrosis of mitral valve, systolic murmur in, 39, 227  
 of coronary arteries, in angina pectoris, 87  
 of valve, in aortic stenosis, 51  
 in mitral stenosis, 39
- Calcium decrease in blood, Q-T interval with, (Fig. 123) 403  
 gluconate, paroxysmal auricular tachycardia and, 193, 194  
 for velocity of blood flow determination, 258
- Camphor, 277
- Cancer of lung, coronary thrombosis, confused with, 118  
 velocity of blood flow in, 259
- Capillary pulse, in aortic insufficiency, 46  
 in thyrotoxic heart disease, 146
- "Cardiac Asthma," hypertension and, 132  
 dyspnea, bronchial dyspnea confused with, 220-223  
 enlargement, electrocardiogram in determination of, 29  
 neurosis. See *Functional heart disease*.  
 output, 252, 266, 267  
 in paroxysmal rapid heart action, 255  
 standstill, quinidine and, 287
- Cardio-pulmonary failure, in deformity of chest, 141
- "Cardio-renal" disease, misuse of term, 249
- Cardio-respiratory, systolic murmur, 228
- Carotid sinus pressure, for anginal attacks, 96  
 in auricular fibrillation, (Fig. 35) 326  
 flutter, (Figs. 30, 35) 322, 326  
 in normal (sinus) tachycardia, (Figs. 35, 70) 326, 358  
 for paroxysmal auricular tachycardia, (Figs. 21, 22, 35) 193, 313-317  
 reflex, 207  
 sinus pauses and, 305  
 sensitivity, syncope in, (Figs. 12, 13) 207, 208, 305, 306
- Cerebral accidents, prognosis of, 209  
 syncope and, 208  
 treatment of, 209, 210  
 aneurysm (ruptured), cerebral hemorrhage confused with, 208  
 emboli, cerebral hemorrhage confused with, 208  
 in coronary thrombosis, 113  
 in mitral stenosis, 209  
 failure, blood pressure fall causing, 106  
 hemorrhage, blood pressure level and, 208, 209  
 conditions confused with, 208  
 coronary thrombosis confused with, 117  
 hypertension and, 130, 131  
 peripheral circulatory failure in, 252  
 spasm, transient hemiplegia and, 209
- Cervical rib, angina pectoris confused with, 91  
 sympathectomy, in angina pectoris, 102, 103  
 veins in, tricuspid stenosis, 55
- Cesarean section, in pregnancy, 243
- Changing first sound, in complete heart block, 365



- Changing first sound in paroxysmal ventricular tachycardia, 202
- Chest deformity, dyspnea in, 225  
pain, in pericarditis, rheumatic, 57  
pulmonary embolism and, 211
- Cheyne-Stokes breathing, aminophyllin for, 262  
caffeine for, 262  
in congestive heart failure, 261  
in coronary thrombosis, 110  
in hypertension, 134  
mechanism of, 261, 262  
morphine for, 262  
paroxysmal dyspnea in, 210
- Cholesterol diet, angina pectoris and, 80, 81
- Chorea, 8, 23  
avertin for, 24  
fever therapy in, 24  
limb weakness in, 24  
in pregnancy, 24, 25  
prognosis of, 22, 23  
treatment of, 24
- Chronic alcoholism, beri-beri heart from, 139  
"myocardial insufficiency," 137  
"myocarditis," meaning of, 136  
paroxysmal auricular fibrillation and, 198
- Circulation, physiology of, 3
- Circus movement, in auricular fibrillation, 327  
flutter, 322  
in paroxysmal auricular tachycardia, 311  
ventricular tachycardia, 202, 347  
quinidine and, 285
- Cirrhosis of liver, pericarditis, constrictive, confused with, 69
- Climate, in angina pectoris, 100  
in prophylaxis of rheumatic fever, 21
- Clubbed fingers, bacterial endocarditis, sub-acute, and, 162  
congenital heart disease and, 162, 172  
cyanosis and, 162  
hereditary type of, 162  
pulmonary infections (chronic) and, 162
- Coarctation of the aorta, blood pressure changes in, 174  
complications of, 174  
hypertension and, 126  
signs of, 174  
x-ray findings in, 174
- Cobra venom, in angina pectoris, 101
- Coffee and premature ventricular beats, 345
- Complete heart block. See *Heart block, third degree.*
- Conduction disturbances in aortic valvular disease, 43
- Congenital heart disease, 170-179  
atrial (auricular) septal defect, 175  
bacterial endocarditis and, 159, 160, 173  
bicuspid valves, 179  
clubbing of fingers and toes in, 172  
congenital heart disease, coarctation of the aorta, 173  
cyanosis in, 171  
dextrocardia, 174, 175  
Eisenmenger complex, 177  
hereditary factor, 170  
idiopathic hypertrophy of heart, 173  
loud murmurs in, 172  
Lutembacher syndrome, 175  
methemoglobinemia confused with, 172  
other congenital abnormalities and, 173-179  
patent ductus arteriosus (Botalli), 177  
foramen ovale, 175  
ventricular septum, 176  
premature beats in, (Fig. 48) 339  
pulmonary stenosis, 177  
tuberculosis and, 172  
retarded growth in, 173  
rheumatic heart disease and, 173  
right aortic arch, 179  
tetralogy of Fallot, 176, 177  
treatment of, 179
- Congestive heart failure, 252-268  
angina pectoris and, 94  
anoxemia of tissues and, 256  
ascites in, 263  
"backward failure" theory, 254  
basal metabolism in, 147  
râles in, 264  
beri-beri heart and, 138-139  
blood volume increase in, 256, 257  
cachexia in, 264  
Cheyne-Stokes breathing in, 261  
clinical picture of, 264, 265  
cough and, 260  
cyanosis in, 264  
digitalis in, 265-268  
dyspnea in, 256  
edema in, 253, 254, 256  
epigastric pain in, 254  
fever in, 262  
"forward failure" theory, 254  
gain of weight in, 265  
gallop rhythm in, 264  
hypertension and, 127, 131-133  
hypoproteinemia and, 279, 280  
icteric index increase in, 257  
jaundice in, 262, 263  
left ventricular, 253, 255  
leukocytosis in, 262  
liver enlargement in, 256  
mechanism of, 254-264  
myxedema heart and, 137  
osmotic pressure of blood and, 256  
paroxysmal nocturnal dyspnea and, 260  
in paroxysmal tachycardia, 190, 191  
349  
peripheral circulatory failure in, 263



- Congestive heart failure, pleural fluid in, 264  
 precipitating causes of, 263  
 in pregnancy, 242  
 psychoses in, 271  
 pulmonary edema in, 256  
   thrombosis and, 211  
 pulsus alternans in, 264  
 right ventricular, 253, 255  
 signs of, 253, 254  
 surgical operations in, 237  
 symptoms of, 253, 254  
 treatment, abdominal paracentesis, 282  
   acid ash diet, 270  
   aims in, 2, 268  
   aminophyllin, 277  
   ammonium salts, 278, 279  
   antipyretics, 280  
   bed rest, 269  
   bladder catheterization, 281  
   blocks under head posts of bed, 269  
   bowels, 271  
   chair (rather than bed) in, 280, 281  
   convalescent care, 284  
   decompression of chest, 291, 292  
   diet, 269  
   digitalis, 271-274. See *Digitalis*.  
   diuretics, 277  
   dorsolumbar sympathectomy, 293  
   fluid intake, 270  
   Karell diet, 269  
   mercuhydrin, 278  
   mercupurin, 278  
   mercurial suppositories, 278  
   mercury, orally, 278  
   morphine, 270, 271  
   oxygen therapy, 282  
   phlebotomy, 281-284  
   plasma intravenously in, 280  
   potassium salts, 277, 279  
   quinidine, 284-291  
   salt restriction, 270  
   salyrgan, 278  
   sedatives, 270  
   semistarvation, 270  
   sociological factors in, 268  
   Southey tubes, 281  
   surgical methods, 291-294  
   theobromine sodium salicylate (diuretin), 277  
   theophyllin (theocin), 277  
   thoracentesis, 281, 282  
   thyroidectomy (total), 292, 293  
   tourniquets, 281, 284  
   urea as diuretic, 279  
   vitamins, 271  
 urinary findings in, 264  
 velocity of blood flow in, 258  
 venous pressure in, 254, 256, 259  
 vital capacity decrease in, 256, 259, 260
- Congestive heart failure, weakness in, 254  
 255  
   without organic disease, (Fig. 59) 349  
   x-ray signs of, 264
- Constitutional type in angina pectoris, 11, 12, 78  
 in pernicious anemia, 11  
 in rheumatic fever, 11, 12
- Constrictive pericarditis, 68-72  
 electrocardiogram in, (Fig. 128) 405, 408  
 rheumatic pericarditis and, 59  
 tricuspid stenosis, resembling, 55  
 tubercular pericarditis and, 62
- Continuous murmur, in arteriovenous fistula, 138
- Convalescence, in coronary thrombosis, 125
- Convalescent care, for congestive heart failure, 284
- Cor pulmonale, acute, 139  
 electrocardiogram in, (Figs. 125-127) 405-407
- Cor pulmonale, chronic, 139  
 diagnosis of, 222
- Coramine, for peripheral circulatory failure, 253
- Coronary artery disease, surgical operations in, 237  
 embolism, in bacterial endocarditis, sub-acute, 163  
 insufficiency, acute, 119  
 ostia occlusion, syphilitic aortitis and, 154  
 sclerosis, angina pectoris, and, 83, 87, 106, 107  
 sinus ligation, for angina pectoris, 104  
 thrombosis, 105-125  
   abdominal findings in, 110, 111  
   absence of cardiac dilation in, 110  
   acute pulmonary edema in, 109  
   Adams-Stokes attacks in, 121  
   aneurysm of ventricle following, 108, 113  
   angina pectoris in its relation to, 105, 106  
   apex impulse in, 110  
   appearance of skin, 109  
   arrhythmias in, 110  
   auricular fibrillation in, 110  
     flutter in, 110  
   blood pressure fall in, 106, 109  
   breathlessness in, 109  
   cerebral embolism in, 113  
   Cheyne-Stokes breathing in, 110  
   clinical features of, 108-114  
   congestive failure following, 113  
   dehydration in, 109, 120  
   differential diagnosis, 115-119  
     acute surgical abdomen, 115  
     angina pectoris, 119  
     arthritis of spine, 118  
     cancer of lung, 118



- Coronary thrombosis, differential diagnosis,  
  cerebral hemorrhage, 117  
  diabetic coma, 116  
  diaphragmatic flutter, 119  
  hernia, 118  
  dissecting aortic aneurysm, 117,  
  118  
  hemorrhage from gastro-intestinal  
  tract, 118  
  herpes zoster, 118  
  incomplete tear of aorta, 118  
  pneumonia, 116  
  pneumothorax, 116  
  pulmonary embolism, 115  
  "spontaneous, interstitial emphy-  
  sema of lungs," 116, 117  
  syphilitic aneurysm, 118  
  disappearance of angina after, 113  
  electrocardiogram in, 111, 112  
  anterior infarction, (Figs. 104-110)  
  386-388  
  lateral lesions, 390, 391  
  posterior infarction, (Figs. 111-115)  
  389-391  
  embolism in, 113  
  etiological factors i., 106  
  extrasystoles in, 110  
  fever in, 109, 110  
  first appearance of angina after, 113  
  gallop rhythm in, 110  
  gallstone colic, confused with, 235  
  gangrene of extremity from 113  
  gastro-intestinal symptoms in, 109  
  glycosuria in, 111  
  heart block in, 110  
  sounds in, 110  
  hemorrhage causing, 106  
  acute, confused with, 213  
  historical notes of, 105  
  leukocytosis in, 109, 110  
  mesenteric infarct in, 113  
  mode of onset, 108  
  oliguria in, 111  
  pain in, 108  
  pancreatitis (acute), confused with, 235  
  paroxysmal auricular fibrillation and,  
  198  
  tachycardia confused with, 191  
  pathological correlations in, 107, 108  
  peptic ulcer (perforated) confused with,  
  235  
  pericarditis and, 63, 110  
  postoperative, (Fig. 145) 426  
  precipitating causes, 107  
  premonitory symptoms of, 108  
  prognosis of, 114, 115  
  pulmonary edema in, 210  
  embolism in, 113  
  findings in, 110  
  renal infarct in, 113  
  rupture of ventricle in, 108, 113
- Coronary thrombosis, sedimentation rate in  
  110  
  shock in, 106, 109  
  splenic infarct in, 113  
  sudden death in, 113  
  "surgical abdomen" confused with, 235  
  from surgical operations, 238  
  sweating in, 109  
  syncope in, 110  
  treatment of, 119-125  
  adrenalin, 120, 121  
  aminophyllin, 122  
  antisyphilitic drugs, 122  
  atropine for ventricular tachycardia  
  121  
  caffeine, 120  
  convalescence in, 125  
  diet, 123  
  digitalis and, 123  
  fluids parenterally, 120  
  insulin, 122, 123  
  intravenous gallbladder dye and, 123  
  magnesium sulfate for ventricular  
  tachycardia, 121, 122  
  morphine, 119  
  oxygen, 120  
  papaverine, 119  
  paredrine, 121  
  plasma, 120  
  quinidine, routine use of, 122  
  for ventricular tachycardia, 121  
  rest in, 123, 124  
  surgical operations and, 123  
  urinary findings in, 111  
  ventricular fibrillation in, 113  
  mural thrombosis in, 108  
  weakness in, 109
- Corrigan pulse, in aortic insufficiency, 46  
  in thyrotoxic heart disease, 146
- Cough, congestive heart failure and, 260  
  in pericarditis, rheumatic, 57
- Coupled beats, from digitalis, 276
- Creatine content of heart muscle, digitalis  
  and, 266
- Cyanosis, argyria confused with, 171, 172  
  clubbed fingers and, 162  
  in congenital heart disease, 171  
  in congestive heart failure, 264  
  in mitral stenosis, 38
- DECHOLIN, for velocity of blood flow de-  
  termination, 258
- Decompression of chest, for congestive heart  
  failure, 291, 292  
  for dysphagia, 291, 292
- Decreased heart sounds, in pericardial ef-  
  fusion, 65
- Defective intraventricular conduction. See  
  *Electrocardiogram in intraventricular block.*
- Deformity of chest, cardiopulmonary failure  
  in, 141



- Deformity of chest, faintness in, 141  
heart failure in, 141
- Dehydration, in coronary thrombosis, 109, 120
- Dextrocardia, electrocardiogram in, 175  
transposition of viscera in, 174
- Diabetes, angina pectoris and, 80  
hypertension and, 131
- Diabetic coma, coronary thrombosis confused with, 116  
peripheral circulatory failure in, 252
- Diaphragmatic flutter, coronary thrombosis confused with, 119  
hernia, angina pectoris confused with, 91  
coronary thrombosis confused with, 118  
"heart noises" in, 117
- Diarrhea, from digitalis, 276  
in thyrotoxic heart disease, 143
- Diastolic murmur, in aortic insufficiency, 45  
stenosis, 52  
apical, in rheumatic carditis (acute), 30  
loud, transmitted to elbow, (Fig. 155) 435  
in mitral stenosis, 32  
in ruptured valve, 138  
thrill, in aortic insufficiency, 44
- Dicoumarin, for bacterial endocarditis, sub-acute, 169
- Diet, in coronary thrombosis, 123  
in rheumatic fever, 17, 19
- Dieulaide's sign of adhesive pericarditis, 74
- Digitalis, action of, 265-268  
for Adams-Stokes disease, 207  
adults' average dose of, 273  
in angina pectoris, 100  
auricular fibrillation and, 265  
caused by, 332  
effect on, (Fig. 36) 328  
auricular standstill and, (Fig. 14) 306, 307  
cardiac output and, 252, 266, 267  
for cerebral accidents, 210  
complete heart block from, (Fig. 42) 333, 363  
conduction effect of, 265  
in congestive heart failure, 265-268, 271-274  
contraction of heart muscle and, 266  
contraindications for use of, 267, 268  
coronary arteries, effect on, 266  
in coronary thrombosis, 123  
coupling, (Figs. 52, 53) 276, 342, 343  
creatine content of heart muscle and, 266  
diarrhea from, 276  
efficiency of heart and, 266  
electrocardiogram and, 266  
electrocardiographic changes from, 276  
extrasystoles and, 265  
extrasystoles from, 276  
heart block from, 276, 358, 359  
muscle injury produced by, 266  
size, effect on, 266
- Digitalis, indications and contraindications 267, 272  
infant's dose, 273  
inhibition of heart from 93  
intramuscular use of, 274, 275  
intravenous use of, 275  
irritability of heart and, 265  
lanata (lanatoside-C), 273  
"maintenance" dose, 274  
margin of safety of, 273  
nausea and, 272, 274  
paroxysmal auricular tachycardia and, (Fig. 26) 194, 195, 199-201, 318, 319  
for paroxysmal dyspnea, 135, 210  
in paroxysmal ventricular tachycardia, 349  
pills or liquid preparations, 272, 273  
potassium content of heart muscle and 266  
P-R interval and, 265  
preventive use of, 267  
prolonged use of, 267  
pulse deficit and, 267  
rectal use of, 274  
refractory period of heart and, 266  
slowing effect of, 265  
S-T changes from, (Fig. 133) 411  
therapeutic dose of, 265  
for thyrotoxic heart disease, 147  
tincture of, 272, 273  
"tonus" effect of, 266  
toxic effects of, 275, 276  
vagal effect of, 265  
yellow vision from, 276
- Digitoxin (Digitaline Nativelle), 273
- Dihydratachysterol, for hypoparathyroidism, (Fig. 123) 403
- Dilatation of heart, in anemia, 29  
in beri-beri heart, 139  
of left auricle, in thyrotoxic heart disease, 145
- Diphtheria, electrocardiogram in, 406, 407  
heart block in, 358
- Disorderly action of the heart. See *Functional heart disease*.
- Dissecting aortic aneurysm, 117, 118  
coronary thrombosis confused with, 117, 118  
limb numbness, pain and paralysis from, 118
- Diuretics, for congestive heart failure, 277  
ill effects of, 280  
redigitalization from, 280
- Diuretin, in angina pectoris, 101
- Dorsal sympathectomy, in angina pectoris, 102
- Dorsolumbar sympathectomy, for congestive heart failure, 293  
for hypertension, 136
- Duration of symptoms, in aortic stenosis, 55  
in mitral stenosis, 55  
in tricuspid stenosis, 55



- Duroziez's sign, in aortic insufficiency, 46  
in thyrotoxic heart disease, 146
- Dysphagia, decompression of chest for, 291, 292  
lusoria, 179  
in mitral stenosis, 38
- Dyspnea, in anemia, 223  
in aneurysm of aorta, 225  
in bronchial disease, 219-223  
in bronchiectasis, 225  
in chest deformity, 225  
in congestive heart failure, 256  
in coronary thrombosis, 109  
in emphysema, 223  
in functional states, 223  
in Hodgkin's disease, 225  
hysterical, 224  
in obesity, 224  
in pneumonia, 225  
in pneumothorax, 225  
in septicemia, 225  
"sighing breathing" causing, 223  
in tuberculosis of lungs, 225  
in tumors of lung, 225
- ECLAMPSIA, hypertension and, 127
- Ectopic beats. See *Premature auricular or ventricular beats*.
- Effort syndrome See *Functional heart disease*.
- Eisenmenger complex, clinical features of, 177
- Electrical alternans, (Figs. 25, 151) 317, 413, 414, 431
- Electrocardiogram, abnormal form of ventricular complex, (Fig. 122) 402  
in angina pectoris, 89, 90  
in anterior infarction, (Figs. 104-110) 386, 388  
in aortic stenosis, 53  
in auricular fibrillation, 35, 327-335  
and extrasystoles, (Fig. 37) 329  
with idioventricular rhythm, (Fig. 42) 329, 333  
paroxysmal, (Fig. 39) 330  
in auricular flutter, 321-327  
hypertrophy ("P" wave), (Figs. 98, 99) 379, 380  
standstill, 307  
in auriculoventricular block, 355-367  
first degree, 355-359  
second degree, 359-362  
third degree, 362-367  
in axis deviation, 374-378  
in beri-beri heart, (Fig. 129) 139, 407  
in bradycardia (normal), 302, 303  
in bundle branch block, 367-373  
(left) with myocardial infarction, (Fig. 145) 424-426  
caution in interpretation of, 412
- Electrocardiogram in complete heart block, 362-367  
in congenital heart disease, 378, 379  
in constrictive pericarditis, (Fig. 128) 405, 408  
in cor pulmonale (acute), (Figs. 125-127), 405-407  
in coronary thrombosis (anterior infarction), (Figs. 104-110) 386-388  
(posterior infarction), (Figs. 111-115) 389-391  
in delayed conduction time, 355-359  
in determination of cardiac enlargement, 29  
in dextrocardia, (Fig. 97) 278, 279  
digitalis and, 266  
in digitalis coupling, (Figs. 52, 53) 342, 343  
digitalis effect on, (Figs. 53, 133) 343, 411  
on S-T complex, (Fig. 133) 411  
in diphtheria, 406, 407  
in emphysema, 405  
in esophageal leads, (Fig. 146) 425-427  
on exercise, in angina, (Fig. 116) 391-397  
fetal heart, 301  
in idioventricular rhythm, (Fig. 11) 305  
on inhalation of 10% oxygen, 391  
in intraventricular block, 373, 374  
(2:1), (Fig. 151) 429-431  
in lateral infarction, 390, 391  
limb potentials, (Figs. 136-138) 416, 419, 420  
low voltage, 381  
in lupus erythematosus disseminatus, 140  
in mitral stenosis, (Figs. 98, 99) 39, 40, 379, 380  
in myxedema, (Fig. 102) 137, 382-384  
in nodal beats, 335-337  
normal, 297-302  
"P" wave, 299-301  
changes, 379, 380  
in paroxysmal auricular tachycardia, 311-321  
with block, (Fig. 28) 320  
nodal tachycardia, (Fig. 46) 337, 338  
ventricular tachycardia, 346-350  
in partial heart block, 359, 362  
in patent ventricular septum, 176  
in pericarditis, constrictive, 70  
rheumatic, 59, (Fig. 121) 401  
position of heart determined by, 416, 417  
in posterior infarction, (Figs. 111-115) 381-391  
in potassium poisoning, (Fig. 124) 404  
precordial (chest) lead, 391-400  
axis deviation, (Figs. 137, 138) 418-420  
bundle branch block, determination of, (Figs. 139, 140) 419-421  
and myocardial infarction, 398  
change in position of exploring electrode, (Fig. 117) 397



- Electrocardiogram, precordial (chest) lead,  
 "extrinsic" deflections, 415  
 historical development of, 414  
 "intrinsic" deflections, 415  
 myocardial infarction, (Figs. 141-146)  
 421-427  
 normal, (Figs. 135, 136) 417-419  
 physiological considerations, 414-416  
 technique of, 301, 302  
 terminology for, 416  
 Wilson's central terminal for, 416  
 in premature auricular beats, 307-311  
   blocked, 309  
   ventricular beats, 337-346  
     interpolated, (Fig. 55) 341, 344  
     multiple, (Fig. 54) 344  
 principles of, 295  
 in pulmonary embolism, 212, 405  
 "QRS" waves (normal), 299-301  
 Q-T interval, normal formulas for, 406  
 quinidine, effect on, 285  
 in rheumatic pericarditis, (Fig. 121) 401,  
 403, 404  
 in scleroderma heart, 140  
 in sino-auricular block, 354, 355  
 in sinus arrhythmia, 303, 304  
   pauses, 304-306  
   tachycardia, 302, 303  
 "ST" deviation, persistence of, (Fig. 120)  
 400  
 T wave changes, 407-409, 412  
   normal, 301  
 in tachycardia (normal), 302, 303  
 in tetralogy of Fallot, (Fig. 95) 376  
 in tricuspid stenosis, 55, 380  
 "U" wave, 300  
 in uremia, (Fig. 124), 404, 405  
 in ventricular fibrillation, 351  
   transient, (Fig. 61) 351, 353  
 in "ventricular flutter," 350, 351  
 in ventricular septal defect, (Fig. 154)  
 434  
 Wilson's terminal, 416  
 in Wolff-Parkinson-White syndrome, 408-  
 411
- Electrocardiographic changes, in coronary  
 thrombosis, 111, 112  
   from digitalis, 276  
   in hypertension, 134, 135  
   in overventilation, 184, 185  
   after paroxysmal auricular tachycardia,  
   192  
   in rheumatic carditis, acute, 28
- Electrocardiography, aims of, 296
- Emboli, to abdominal organs, 212  
 in bacterial endocarditis, subacute, 162  
 in coronary thrombosis, 113  
 in paroxysmal auricular fibrillation, 199  
 peripheral, 212, 213  
   in mitral stenosis, 38  
 quinidine and, 286-288
- Emergencies, acute cardiovascular, 204-213
- Emphysema, heart, 223  
 dyspnea in, 223  
 electrocardiogram in, 405  
 velocity of blood flow in, 259
- Endocarditis lente. See *Bacterial endo-  
 carditis, subacute.*  
 non-bacterial, 156. See *Rheumatic fever.*  
 in lupus erythematosus disseminatus, 140
- Endocrine glands, in relation to other dis-  
 eases, 25, 26  
 rheumatic fever and, 25
- Ephedrine sulfate, for Adams-Stokes dis-  
 ease, 205  
 for carotid sinus sensitivity, 208  
 in complete heart block, 367  
 for syncope, 205
- Epigastric pain, in congestive heart failure,  
 254
- Epilepsy, syncope in, 206
- Epistaxis, in rheumatic fever, 10
- Erosion of bone, in syphilitic aneurysm of  
 aorta, 153
- Erysipelas, first degree heart block in, (Fig.  
 70) 358
- Erythrol tetranitrate, in angina pectoris, 101
- Esophageal compression, in mitral stenosis,  
 38, 39  
 leads, in diagnosis of posterior infarction,  
 (Fig. 146) 427
- Ether, for velocity of blood flow determin-  
 ation, 258
- Ewart's sign in pericarditis, rheumatic, 85
- Exercise, systolic murmur and, 230  
 test, for angina pectoris, 90  
   for presystolic murmur, 32
- Exophthalmos, in hyperthyroidism, 151
- Exposure to wetness and chilliness, rheu-  
 matic fever and, 20
- Extraction of teeth, bacterial endocarditis,  
 subacute, and, 161
- Extrasystoles, 182, 183. See *Functional  
 heart disease, Premature auricular or  
 ventricular beats.*  
 in coronary thrombosis, 110  
 from digitalis, 276  
 mechanism of, 182  
 quinidine in, 291
- FAINTNESS, in deformity of chest, 141
- Familial factor, in rheumatic fever, 11
- Fever, in congestive heart failure, 262  
 in coronary thrombosis, 109, 110  
 increase of basal metabolism in, 147  
 in paroxysmal auricular tachycardia, 191  
 in potential hypertension, 233  
 prolonged in rheumatic fever, 14  
 in pulmonary embolism, 211  
 systolic murmur in, 229  
 velocity of blood flow and, 146



- Fever therapy, in bacterial endocarditis, sub-  
acute, 167, 168  
in chorea, 24  
"Fever unknown origin," coronary throm-  
bosis confused with, (Fig. 105) 385  
First sound, accentuation of, 32  
in anemia, 32  
in cardiac neurosis, 32  
in hyperthyroidism, 32  
in mitral stenosis, 32  
in complete heart block, 365  
phonocardiogram of, (Fig. 152)  
432  
in first degree heart block, 359  
variations in, P-R interval and, (Fig.  
152) 431, 432  
Flushing of neck, in potential hypertension,  
233  
"Focus of infection," rheumatic fever and,  
7  
"Forward failure" theory, congestive heart  
failure, 254  
Freckling, hypertension and, 128  
rheumatic type and, 12  
Friction rub, and pericardial effusion, 65  
in pericarditis, rheumatic, 57, 58  
Functional heart disease, 180-187  
angina pectoris confused with, 91  
arrhythmias in, 181  
definition of, 180  
hyperthyroidism confused with, 184  
prognosis of, 187  
symptoms of, 183, 184  
synonyms of, 5  
systolic murmur in, 180, 181  
tuberculosis confused with, 184  
Functional tests of heart, inadequacy of, 6  
"Funnel chest," 225
- GALLBLADDER disease, angina pectoris con-  
fused with, 91  
Gallop rhythm, 133, 134  
in beri-beri heart, 139  
in congestive heart failure, 264  
in coronary thrombosis, 110  
diastolic, phonocardiogram of, (Fig. 150)  
429, 430  
in first degree heart block, 359  
hypertension and, 132, 133  
mid-systolic, 134  
normal, phonocardiogram of, (Fig.  
149) 429, 430  
presystolic, 133  
protodiastolic, 133  
in rheumatic carditis (acute), 28  
in scleroderma heart, 140  
Gallstone colic, coronary thrombosis con-  
fused with, 235  
Gangrene of extremity from coronary  
thrombosis, 113  
Gastro-intestinal symptoms in coronary  
thrombosis, 109  
Glycosuria, in coronary thrombosis, 111  
in thyrotoxic heart disease, 143  
Gout, angina pectoris and, 81  
Graham-Steell murmur, 47  
Gray hair, in arteriosclerosis, 144  
in asthma, 144  
in pernicious anemia, 144  
in thyrotoxic heart disease, 144  
"Growing pains," 8  
Gumma of heart, heart block in, 155
- HAIR, early graying of, arteriosclerosis and,  
144  
asthma and, 144  
hyperthyroidism and, 144  
pernicious anemia and, 144  
Hamman's disease, 116, 117  
Heart block, atropine effect on, (Fig. 75) 362  
causes of, 358, 359  
in coronary thrombosis, 110  
from digitalis, 276, 358, 359  
first degree, in erysipelas, (Fig. 70) 358  
first heart sound in, 359  
gallop rhythm in, 359  
paroxysmal tachycardia confused  
with, (Fig. 70) 358  
in rheumatic fever (Figs. 66, 68)  
355, 356  
valvular disease, (Fig. 67) 356  
in gumma of heart, 155  
in hyperthyroidism, 359  
in patent ventricular septum, 176  
in pericarditis, rheumatic, 59  
in rheumatic carditis, (acute), 28  
fever, 358, 359  
second degree, (Figs. 71, 72, 73) 359-  
362  
diagnosis of, 361, 362  
regular rhythm in, (Fig. 74) 361  
syncope in, (Fig. 74) 361  
treatment of, 362  
Wencheback phenomenon in, (Fig.  
71) 359, 361  
third degree, (Figs. 76-82) 363-367  
Adams-Stokes attacks in, 364, 365  
adrenalin in, 366, 367  
bedside diagnosis of, 365, 366  
in childhood, (Fig. 78) 363, 364  
clinical features of, 364-367  
congenital, (Figs. 78, 79) 363, 364  
from digitalis, (Fig. 42) 333, 363  
ephedrine in, 367  
first heart sound in, 365  
hypertension and, 127  
irregularity of heart in (Fig. 78) 364  
paroxysmal ventricular tachy-  
cardia in, (Fig. 62) 352, 365  
sudden death in, 92



- Heart block, third degree, syncope in, (Fig. 77) 353, 365  
 treatment of, 366, 367  
 disease, allergic state and, 219, 220  
 forms of, 4  
 failure. See *Congestive heart failure*.  
 in acute nephritis, 139  
 in bacterial endocarditis, subacute, 163  
 in deformity of chest, 141  
 in mitral insufficiency (organic), 227  
 syphilitic aneurysm of aorta and, 153  
 "noises," 117  
 size in angina pectoris, 87  
 sounds, 3, 4. See also *Phonocardiography*.  
 in coronary thrombosis, 110  
 hyperactive. See *Hyperactive heart sounds*.  
 normal, (Fig. 147) 428, 429
- Hematuria, in bacterial endocarditis, subacute, 161, 162
- Hemiplegia (transient), cerebral spasm and, 209
- Hemoptysis, in mitral stenosis, 38, 39  
 pulmonary embolism and, 211
- Hemorrhage, acute, coronary thrombosis  
 confused with, 213  
 causing coronary thrombosis, 106  
 from gastro-intestinal tract, coronary  
 thrombosis confused with, 118  
 massive, syncope in, 206  
 peripheral circulatory failure in, 252
- Hemorrhagic pericardial fluid, in tumors of  
 heart, 140
- Heparin, for bacterial endocarditis, subacute,  
 169
- Heredity, angina pectoris and, 77, 78  
 in hypertension, 128
- Herpes zoster, angina pectoris confused  
 with, 91  
 coronary thrombosis confused with, 118
- His bundle, 297, 298
- Hoarseness, in mitral stenosis, 38
- Hodgkin's disease, dyspnea in, 225
- Hyperactive heart sounds, 145  
 in potential hypertension, 233  
 in rheumatic fever, 9  
 in thyrotoxic heart disease, 145
- Hypercholesteremia, angina pectoris and, 78,  
 80
- Hypertension, aberrant renal artery and, 136  
 adrenal tumors and, 127, 136  
 aortic insufficiency and, 127, 134  
 arteriosclerosis and, 129  
 basal metabolism increase in, 147  
 "cardiac asthma" and, 132  
 cardiac murmurs in, 134  
 cerebral hemorrhage and, 130, 131  
 chronic nephritis and, 127, 131  
 Cheyne-Stokes breathing in, 134  
 clinical course of, 130-133  
 coarctation of aorta and, 126
- Hypertension, congestive failure and, 127,  
 131-133  
 diabetes and, 131  
 dorsolumbar sympathectomy for, 136  
 eclampsia and, 127  
 electrocardiographic changes in, 134, 135  
 essential, 128  
 etiological factors in, 128  
 freckling and, 128  
 gallop rhythm and, 132, 133  
 heart block (complete) and, 127  
 heredity in, 128  
 intracranial hemorrhage and, 127  
 malignant, 135  
 mitral insufficiency in, 134  
 stenosis and, 41, 42  
 nephrectomy (unilateral) and, 128  
 in nephritis, acute, 127  
 nocturnal dyspnea and, 132  
 "paroxysmal cardiac dyspnea" and, 132  
 pituitary basophilism and, 127  
 polycystic kidneys and, 127  
 polycythemia and, 127  
 potential, 128  
 prognosis of, 135  
 prostatic obstruction and, 126  
 pulmonary congestion in, 134  
 pulsus alternans and, 132, 133  
 pyelonephritis and, 127, 136  
 reflexes from heart and, 127  
 relative insufficiency of valves in, 134  
 renal ischemia (Goldblatt mechanism),  
 and, 128  
 retinal changes in, 129  
 rheumatic fever and, 129  
 surgical operations in, 237  
 treatment of, 136  
 systolic murmur in, 229  
 vascular vulnerability and, 128
- Hypertensive encephalopathy, 135  
 heart disease, 126-136  
 failure, treatment of, 135, 136  
 sympathectomy (dorsolumbar) for,  
 293
- Hyperthyroidism, 142-152. See also *Thyro-  
 toxic heart disease*.  
 beri-beri heart and, 139  
 clinical features of, 142-147  
 exophthalmos in, 151  
 functional heart disease confused with, 184  
 gray hair and, 144  
 heart block in, 359  
 paroxysmal auricular fibrillation and, 198  
 pituitary gland and, 151  
 systolic murmur in, 229
- Hypertrophy of heart in pericarditis, chronic,  
 non-constrictive, 73
- Hypoglycemia, angina pectoris and, 86, 98
- Hypoparathyroidism, dehydrotachysterol for,  
 (Fig. 123) 413  
 Q-T interval in, (Fig. 123) 403, 406



- Hypoproteinemia, congestive heart failure and, 279, 280  
 in pericarditis, constrictive, 70  
 in tricuspid stenosis, 55  
 Hysterical dyspnea, 224
- ICTERIC index, increase in congestive heart failure, 257  
 Idiopathic hypertrophy of the heart, glycogen deposits causing, 178  
 Inequality of pupils and pulses, in syphilitic aneurysm of aorta, 153  
 Infantile paralysis, compared to rheumatic fever, 13  
 Infections, peripheral circulatory failure in, 252  
 Infectious arthritis, rheumatic fever confused with, 15  
 (Inhibition of heart, causing sudden death, 92, 93  
   from digitalis, 93  
   from quinidine, 93)  
 Insulin, in coronary thrombosis, 122, 123  
 "Intermittent pulse," 337, 338  
 Intracranial hemorrhage, hypertension and, 127  
 Intraventricular block, (Figs. 91, 92) 373, 374  
 Ipecac, for paroxysmal auricular tachycardia, 192
- JAUNDICE, bradycardia in, 303  
 in congestive heart failure, 262, 263  
 in pulmonary infarction, 211  
 Jews, angina pectoris and, 79  
 Jugular pulse, in paroxysmal ventricular tachycardia, 202
- KARELL diet, in angina pectoris, 98  
 in congestive heart failure, 269  
 Keith-Flack node, 297
- LEAD poisoning, angina pectoris and, 81  
 Left hand disability, in angina pectoris, 91  
 shoulder pain in angina pectoris, 91  
 ventricular heart failure, 253, 255  
 Leukemia, basal metabolism increase in, 147  
 Leukocytosis, in congestive heart failure, 262  
 in coronary thrombosis, 109, 110  
 in paroxysmal auricular tachycardia, 191  
 in pulmonary embolism, 211  
 in rheumatic fever, 15  
 Leukopenia, in lupus erythematosus disseminatus, 141  
 Libman-Sacks disease, 140-141  
 Liver enlargement, in congestive heart failure, 256  
 in pericardial effusion, 65  
 Liver enlargement, in pericarditis, constrictive, 69  
 in tricuspid stenosis, 54  
 pulsation, in tricuspid insufficiency, 53, 54  
 Loud murmurs, in congenital heart disease, 172  
 systolic murmur (aortic), transmitted to elbow, (Fig. 153) 432  
 Low voltage of QRS complex, conditions with, 382-386  
 in myxedema, (Fig. 102) 382-384  
 in normal hearts, (Fig. 103) 383  
 Lugol's solution, for thyroid heart disease 147  
 Lung tumors, dyspnea in, 225  
 Lupus erythematosus disseminatus, 140  
 albuminuria in, 141  
 electrocardiogram in, 140  
 endocarditis, non-bacterial in, 140  
 leukopenia in, 141  
 menstruation and, 140  
 pericarditis and, 64, 140  
 rheumatic fever confused with, 15, 16  
 x-ray treatment for, 141  
 Lutembacher syndrome, 175, 176
- MACHINERY murmur, in patent ductus arteriosus, 177  
 phonocardiogram of, (Fig. 156) 436  
 Magnesium sulfate, in paroxysmal auricular tachycardia, (Fig. 27) 194, 319  
 for paroxysmal ventricular tachycardia, 121, 122, 203, 350  
 Malignant arteriosclerosis, syphilis and, 152  
 hypertension, 135, 136  
 "nephrosclerosis," 135, 136  
 "Masked thyrocardiacs," 143  
 Mecholyl, in paroxysmal auricular tachycardia, (Fig. 25) 193, 317, 318  
 Mediastinopericarditis, 72-76. See *Pericarditis, chronic, non-constrictive*.  
 Medicolegal aspects of heart disease, 214-219  
 Menstruation, lupus erythematosus disseminatus and, 140  
 Mercuhydrin, for congestive heart failure, 278  
 Mercupurin, for congestive heart failure, 278  
 Mercurial suppositories, for congestive heart failure, 278  
 Mesenteric infarct, in coronary thrombosis, 113  
 Methemoglobinemia, congenital heart disease confused with, 172  
 methylene blue in treatment of, 172  
 Metrazol, for Adams-Stokes disease, 207  
 Military considerations of neurocirculatory asthenia, 187  
 Mitral insufficiency, in hypertension, 134  
 organic, evidence of existence of, 31, 39



- Mitral insufficiency, organic, heart failure in, 227  
 relative, 134  
 rheumatic, 30-32  
 stenosis, 32-42  
   accentuation of first sound in, 32  
     of pulmonary second sound in, 32  
   age at death in, 55  
   aphonia in, 38  
   auricular (left) dilatation in, 39  
   axis deviation (right) in, (Fig. 94) 40, 376  
   blood pressure and, 41, 42  
   bronchial compression in, 38, 39  
     veins in, 39  
   bronchiogenic carcinoma confused with, 38  
   calcification of valve in, 39  
   cerebral embolism in, 209  
   chronicity of failure in, 38  
   common signs of heart failure in, 40  
   cyanosis in, 38  
   development of auricular fibrillation in, 38  
     of murmurs in, 32, 33  
   diagnosis of, 39  
   diagnostic importance of auscultation in, 37  
   diastolic murmur in, 32  
   without diastolic murmur, 37  
   duration of symptoms in, 55  
   dysphagia in, 38  
   electrocardiogram in, (Figs. 98, 99) 39, 40, 379, 380  
   emboli (peripheral) in, 38  
   esophageal compression in, 38, 39  
   favorable effect of hypertension in, 42  
   general features of, 38  
   hemoptysis in, 38, 39  
     mechanism of, in, 39  
   hoarseness in, 38  
   hypertension and, 41, 42  
   paroxysmal auricular fibrillation and, 198  
   patent ductus arteriosus distinguished from, 39  
   presystolic murmur in, 32  
   pulmonary infarction in, 38  
   "P" wave changes in, 39, 40  
   rheumatic carditis (acute), confused with, 30  
   systolic murmurs in, 40  
   thrill (diastolic) in, 37, 38  
   thyrotoxic heart disease confused with, 145  
   tuberculosis confused with, 38  
   vascular vulnerability and, 42  
   valvulotomy for, 291  
   x-ray in diagnosis of, 39  
 Morphine, in angina pectoris, 96  
   for Cheyne-Stokes breathing, 262  
   Morphine in congestive heart failure, 270, 271  
     in coronary thrombosis, 119  
     for paroxysmal dyspnea, 135  
 Mural thrombosis, in beri-beri heart, 139  
 Murmurs, transmission of, significance of, 435, 436  
 Myxedema heart, 137  
   angina and, 81, 137  
   congestive heart failure and, 137  
   electrocardiogram in, 137  
   thyroid medication for, 137  
 Myxoma of auricle, 140  
 NEOPLASM of heart, hemorrhagic pericardial effusion in, 140  
 Neosalvarsan, for bacterial endocarditis, subacute, 167  
 Nephrectomy (unilateral), hypertension and, 128  
 Nephritis, acute, heart failure in, 139  
   hypertension in, 127  
   chronic, from bacterial endocarditis (subacute), 163  
   hypertension and, 127, 131  
   pericarditis and, 62, 63  
   surgical operations and, 237  
   diuretics and, 279  
 Nervous heart. See *Functional heart disease*.  
 Neurocirculatory asthenia. 180-187. See *Functional heart disease*.  
   constitutional factor, 185  
   military considerations of, 187  
   physical findings in, 186  
   precipitating factors, 185  
   premature ventricular beat in, (Fig. 47) 338  
   prognosis of, 187  
   symptoms of, 186  
   trauma and, 216  
   treatment of, 186, 187  
 Nitroglycerin, in angina pectoris, 95  
   paroxysmal auricular tachycardia and, 194  
 Nitroglycerin workers, angina pectoris and, 83  
 Nocturnal dyspnea, hypertension and, 132  
 Nodal beats, (Fig. 45) 335-337  
   rhythm, (Figs. 43, 44) 335, 337  
 Nodules, in rheumatic fever, 5  
 Non-constrictive, pericarditis, chronic, 72-76  
 Non-valvular heart disease, 137  
 Normal heart, congestive failure from arrhythmias in, (Fig. 59) 349  
   paroxysmal auricular fibrillation and, 198  
 Nose-bleeds, in rheumatic fever, 10  
 OBESITY, dyspnea and, 224  
   prognosis in angina pectoris and, 94  
   vital capacity of lungs, 88, 89



- Obstetrics, risk of cardiacs and, 239-244.  
See also *Pregnancy*.
- Ocular pressure, in paroxysmal auricular tachycardia, (Fig. 23) 193, 314, 317
- Oculocardiac reflex, 193
- Omentopexy, for angina pectoris, 104
- Operations (especially on the lungs), paroxysmal auricular fibrillation and, 198
- Operative mortality, in thyrotoxic heart disease, 149, 151
- Osmotic pressure of blood, congestive heart failure and, 256
- Ouabain, 273
- Overventilation, electrocardiographic changes in, 184, 185  
tetany in, 184, 223
- Oxygen therapy, for coronary thrombosis, 120  
for congestive heart failure, 282
- PAGET'S disease, angina pectoris and, 81
- Pain, in coronary thrombosis, 108
- Painful finger tips, in bacterial endocarditis, subacute, 161, 162
- Pancreatitis, acute, coronary thrombosis confused with, 235
- Papaverine, in coronary thrombosis, 119
- Paradoxical embolism, septal defects and, 175
- Paredrine, in coronary thrombosis, 121  
in peripheral circulatory failure, 253
- Paroxysmal auricular fibrillation, 198-201  
"chronic myocarditis" and, 198  
coronary thrombosis and, 198  
diagnosis of, 198, 199  
emboli in, 199  
hypertensive heart disease and, 198  
hyperthyroidism and, 198  
mitral stenosis and, 198  
normal heart and, 198  
operations (especially on the lungs) and, 198  
pneumonia and, 198  
quinidine for, 199-201  
rheumatic fever and, 198  
symptoms of, 199  
treatment of, 199-201  
in Wolff-Parkinson-White syndrome, 409
- flutter, 196-198  
digitalis for, 197  
quinidine for, 197  
treatment of, 197, 198
- tachycardia, 311-321  
carotid sinus effect on, (Figs. 21, 22, 35) 313-317  
changes in dynamics of circulation in, 191  
circus movement in, 311  
congestive failure in, 190, 191  
constancy of rate of, 189
- Paroxysmal auricular tachycardia, coronary thrombosis confused with, 191, 192  
digitalis in treatment of, (Fig. 26) 199-201, 318, 319  
during anesthesia, 192  
electrocardiographic abnormalities following, 192  
fever in, 191  
heart block (first degree) confused with, (Fig. 70) 358  
leukocytosis in, 191  
magnesium sulfate in, (Fig. 27) 319  
mecholyl in, (Fig. 25) 317, 318  
ocular pressure in, (Fig. 23) 314, 317  
peripheral thrombosis in, 190  
physical findings in, 190, 191  
precipitating causes of, 190  
prevention of attacks, 194, 195  
pulsus alternans in, 413  
quinidine in, (Fig. 24) 314, 318, 319  
regularity of beats in, 189  
symptoms of, 189, 190  
treatment of, 192-196  
unusual type, with block, (Fig. 28) 195, 196, 319, 320  
in Wolff-Parkinson-White syndrome, 408, 409
- cardiac dyspnea, 210  
aminophyllin (intravenously) for, 135  
Cheyne-Stokes breathing in, 210  
digitalis for, 135  
hypertension and, 132  
morphia for, 135  
phlebotomy for, 135  
tourniquets for, 135  
treatment of, 135, 210
- nocturnal dyspnea, and congestive heart failure, 260  
mechanism of, 260
- nodal tachycardia, (Fig. 46) 337, 338
- rapid heart action, 188-204  
angina pectoris and, 81, 82, 83  
cardiac output in, 255  
pulmonary edema in, 210, 213  
syncope in, 190, 205  
types of, 188  
vagal stimulation in, (Fig. 35) 203, 204
- ventricular tachycardia, 201-203  
atropine in, 349  
bedside diagnosis of, 348  
changing first heart sound in, 202  
diagnosis of, 201, 202  
digitalis in, 349  
on effort, 345  
electrocardiographic abnormalities following, 347, 348  
in heart block (complete), (Fig. 62) 352, 365  
irregularities in rhythm in, (Figs. 53, 57, 58) 202, 346-348



- Paroxysmal ventricular tachycardia, jugular pulse in, 202  
 magnesium sulfate in, 350  
 mechanism of, 202, 347  
 in normal heart, (Fig. 59) 348, 349  
 quinidine in, (Figs. 59, 132) 291, 349, 410  
 treatment of, 202, 203, 349, 350  
 in Wolff-Parkinson-White syndrome, (Fig. 132) 409
- Patent ductus arteriosus, bacterial endocarditis in, 160, 163, 178  
 blood pressure in, 177  
 clinical features of, 177  
 electrocardiogram in, 178  
 machinery murmur in, 177  
 mitral stenosis confused with, 39  
 stunted growth in, 178  
 surgical division of, 178  
 x-ray findings in, 177, 178
- foramen ovale, 175  
 ventricular septum, 176  
   bacterial endocarditis and, 176  
   clinical features of, 176  
   electrocardiogram in, 176  
   heart block in, 176
- Pectoral muscle, implantation for angina pectoris, 104
- Penicillin, for bacterial endocarditis, acute, 158, 159  
   subacute, 168, 169
- Peptic ulcer, angina pectoris and, 99  
   perforated, coronary thrombosis confused with, 235
- Pericardial diseases, 56-76  
   effusion, 64-66  
     decreased heart sounds in, 65  
     frictional rub and, 65  
     heart shape in, 65  
     hemorrhagic, and neoplasm, 68  
     liver enlargement with, 65  
     pulsus paradoxus in, 65  
     signs of, 65  
     tapping of, 66-68  
     x-ray signs of, 66  
   friction, in coronary thrombosis, 110  
   rheumatic carditis, acute, and, 30  
   tap, 66-68
- Pericarditis, acute, "surgical abdomen" confused with, 234  
   chronic nephritis and, 62, 63  
   chronic, non-constrictive, 72-76  
     etiology of, 72, 73  
     hypertrophy of heart in, 73  
     signs of, 73-75  
     treatment of, 75  
     valvular disease confused with, 75
- constrictive, 68-72  
   ascites in, 69  
   cirrhosis of liver confused with, 69  
   decreased cardiac output in, 68, 70
- Pericarditis, constrictive, Delorme's operation for, 71  
   diagnosis of, 69  
   electrocardiogram in, 70  
   etiology of, 68, 69  
   hypoproteinemia in, 70  
   liver enlargement in, 69  
   prognosis in, 72  
   pulsus paradoxus in, 69  
   resection of pericardium for, 71, 72  
   symptoms of, 69  
   treatment of, 71  
   tricuspid stenosis confused with, 71  
   venous pressure in, 68, 70  
   x-ray findings in, 70, 71
- coronary thrombosis and, 63  
 lupus erythematosus disseminatus and, 64  
   140
- miscellaneous forms of, 64  
 in pneumonia, 61  
 rheumatic, 56-61  
   abdominal pain in, 57  
   aortic insufficiency confused with, 58  
   chest pain in, 57  
   complete recovery from, 60  
   constrictive pericarditis and, 59  
   cough in, 57  
   diagnosis of, 57  
   electrocardiogram in, 59, (Fig. 121) 401  
   Ewart's sign in, 58  
   friction rub in, 57, 58  
   heart block in, 59  
   importance of valves in recovery from, 60  
   pneumonia confused with, 58  
   prognosis of, 59, 60  
   symptoms of, 57  
   treatment of, 60
- tuberculous, 62  
   constrictive pericarditis and, 62  
   large effusions in, 62
- Pericardium, empyema of, 61
- Peripheral circulatory failure, 252, 253  
   in anesthesia, 252  
   in burns, 252  
   caffeine sodium benzoate for, 253  
   in cerebral hemorrhage, 252  
   in congestive heart failure, 263  
   coramine for, 253  
   in diabetic coma, 252  
   in hemorrhage, 252  
   in infections, 252  
   paredrine for, 253  
   plasma for, 253  
   in pneumonia, 252  
   signs of, 252, 253  
   in surgical operations, 252  
   in trauma, 252  
   treatment of, 252, 253
- neuritis, beri-beri heart and, 138, 139  
 signs, of aortic insufficiency, 46



- Peripheral signs in other conditions, 46  
 veins, pulmonary embolism and, 211
- Pernicious anemia, angina pectoris and, 82  
 cardiovascular accidents and, 82  
 constitutional type in, 11  
 gray hair and, 144
- "Perpetual arrhythmia," 198. See *Auricular fibrillation*.
- Persistent ostium primum. See *Atrial septal defect*.  
 secundum. See *Atrial septal defect*.
- Petechiae, in bacterial endocarditis, sub-acute, 161
- Phlebotomy, for cerebral accidents, 210  
 for congenital heart disease, 179  
 for congestive heart failure, 281-284  
 for paroxysmal dyspnea, 135, 210
- Phonocardiography, 427-437  
 auricular fibrillation, (Fig. 148) 429  
 changing first sound in complete block, (Fig. 152) 432  
 diastolic gallop, (Fig. 150) 429, 430  
 murmur (loud) transmitted to elbow, (Fig. 155) 435  
 machinery murmur of patent ductus, (Fig. 156) 436  
 mid-systolic (normal) gallop, (Fig. 149) 429, 430  
 normal heart sounds, (Fig. 147) 428, 429  
 systolic murmur produced by fever, (Fig. 157) 437  
 loud, transmitted to elbow, (Fig. 153) 432  
 of Roger's disease transmitted to elbow (Fig. 154) 434  
 uses of, 428  
 ventricular alternation, (Fig. 151) 429, 430
- Phyllicin, in angina pectoris, 101
- Pick's disease, 73, 74
- "Pistol shot," in aortic insufficiency, 46  
 in thyrotoxic heart disease, 146
- Pituitary basophilism, hypertension and, 127  
 gland, hyperthyroidism and, 151
- Plasma, for congestive heart failure, 280  
 in coronary thrombosis, 120  
 for peripheral circulatory failure, 253
- Plateau pulse in aortic stenosis, 53
- Pleural fluid, in congestive heart failure, 264
- Pneumonia, coronary thrombosis confused with, 116  
 dyspnea in, 225  
 paroxysmal auricular fibrillation and, 198  
 pericarditis in, 61  
 rheumatic, confused with, 58  
 peripheral circulatory failure in, 252  
 velocity of blood flow in, 259
- Pneumothorax, coronary thrombosis confused with, 116  
 dyspnea in, 225
- Poliomyelitis, tonsillectomy and, 19
- Polycystic kidneys, hypertension and, 127
- Polycythemia, angina pectoris and, 81  
 cerebral hemorrhage confused with, 208  
 hypertension and, 127  
 in tetralogy of Fallot 176  
 in tricuspid stenosis, 54
- Polygram, in auricular fibrillation, 35
- Postoperative bacterial endocarditis, sub-acute, 161  
 pulmonary embolism, 211
- Potassium chloride, for congestive heart failure, 279  
 content of heart muscle, digitalis and, 266,  
 iodide, in angina pectoris, 99, 100  
 poisoning, electrocardiogram in, (Fig. 124) 404, 405  
 salts for premature beats, 310, 311, 345
- Potential heart disease, 26-30  
 hypertension, fever in, 233  
 flushing of neck in, 233  
 hyperactive heart sounds in, 233  
 systolic murmur in, 233
- Precordial pain in rheumatic fever, 14
- Pregnancy, abortion, indications for, in  
 cardiacs, 239-244  
 auricular fibrillation and, 240  
 beri-beri heart in, 139  
 caesarean section in, 243  
 chorea and, 24, 25  
 congestive heart failure in, 242  
 contraindications for, 239  
 maternal mortality of cardiacs with, 240  
 243  
 pulmonary edema in, 242  
 ultimate effect of, in cardiacs, 243, 244  
 vital capacity of lungs in, 242
- Premature auricular beats, 307-311  
 beats, potassium salts for, 310, 311  
 ventricular beats, atropine for, 345  
 coffee and, 345  
 compensatory pause in, 339  
 in congenital heart disease, (Fig. 48) 339  
 on effort, 345  
 inaudible, 338, 339  
 in neurocirculatory asthenia, (Fig. 47) 338  
 potassium salts for, 345  
 quinidine for, 345  
 symptoms of, 344  
 tobacco and, 345  
 treatment of, 345
- Presystolic murmur, amyl nitrite test for, 32  
 exercise test for, 32  
 methods of detection of, 32  
 in mitral stenosis, 32
- Prognosis (in heart disease), 244-251  
 "accidents" of heart disease, 249-251  
 in angina pectoris, 92-95, 251  
 in aortic disease, 248  
 arteriovenous fistula, 249  
 auricular fibrillation and, 245



- Prognosis (in heart disease), bacterial endocarditis, 250  
 beri-beri heart, 249  
 bronchial dyspnea, role of, 246  
 bundle branch block, 248  
 cardiac enlargement, 245  
 Cheyne-Stokes breathing, 249  
 chronic nephritis, 249  
 constrictive pericarditis, 249  
 coronary thrombosis, 114, 115, 251  
 emboli, 250  
 functional dyspnea, role of, 247  
 gallop rhythm, 248  
 heart block, 250  
   rate, 245  
 hypertension in mitral stenosis, 248  
 immediate in contrast to ultimate, 244, 245  
 infection, role of, 246  
 mitral disease, 248  
 nephritis, acute, 249  
 nocturnal dyspnea, 249  
 obesity, 247  
 paroxysmal dyspnea, 248  
   rapid heart action, 249, 250  
 pulsus alternans, 248  
 thyroid heart disease, 249  
 toxemia of pregnancy, 249  
 tricuspid disease, 248  
 Propadrine, for Adams-Stokes disease, 206  
 Prostatic obstruction, hypertension and, 126  
 Prostigmine methyl-sulfate, for paroxysmal auricular tachycardia, 194  
 Psychoses, in congestive heart failure, 271  
 Pulmonary arteriosclerosis in mitral stenosis, 129  
   congestion, in hypertension, 134  
   edema, acute, 210  
   in coronary thrombosis, 109  
   velocity of blood flow in, 259  
   in congestive heart failure, 256  
   in coronary thrombosis, 210  
   in paroxysmal rapid heart action, 210, 213  
   in pregnancy, 242  
   in pulmonary embolism, 210  
 embolism, 211, 212  
   auricular thrombi and, 211  
   in bacterial endocarditis, subacute, 163  
   cardiac findings in, 211  
   chest pain and, 211  
   in coronary thrombosis, 113  
   coronary thrombosis confused with, 115  
   edema in, 210  
   electrocardiogram in, (Figs. 125-127), 212, 405-407  
   fever in, 211  
   hemoptysis and, 211  
   infarction and, 211  
   leukocytosis in, 211  
   peripheral veins and, 211  
   postoperative, 211  
 Pulmonary embolism, shock in, 211  
   infarction, jaundice in, 211  
   in mitral stenosis, 38  
   pulmonary embolism and, 211  
   infections (chronic), clubbed fingers and, 162  
   stenosis, axis deviation (right) in, (Fig. 95) 376  
   congenital, 177  
   thrombosis, congestive heart failure and, 211  
   tuberculosis, congenital heart disease and, 172  
 Pulsating veins, in tricuspid insufficiency, 53  
 54  
 Pulse deficit, in auricular fibrillation, 34  
   brachigram showing, (Fig. 52) 342  
   digitalis and, 267  
   pressure, in aortic insufficiency, 46  
   in arteriovenous fistula, 138  
   in beri-beri heart, 139  
 Pulsus alternans, (Fig. 134) 132, 133, 412-414  
   alternation of apex impulse in, 413  
   of heart sounds in, (Fig. 151) 413, 429  
   in congestive heart failure, 264  
   hypertension and, 132, 133  
   in paroxysmal tachycardia, 413  
   pseudo, extrasystoles producing, 412  
   paradoxus, in pericardial effusion, 65  
   in pericarditis, constrictive, 69  
 Purkinje fibers, 298  
 Pyelonephritis, hypertension and, 127, 136
- Q-T INTERVAL, calcium in blood and, (Fig. 123) 403  
   in hypoparathyroidism, (Fig. 123) 403, 406  
   normal standards for, 406
- Quinidine, in angina pectoris, 101  
   in auricular fibrillation, (Figs. 37, 40, 41) 289, 290, 329, 331, 335  
   without heart disease, 288  
   flutter, (Fig. 29) 321, 327  
   auricular standstill and, (Fig. 15) 307  
   cardiac standstill and, 287  
   circus movement and, 285  
   in congestive heart failure, 284-291  
   in coronary thrombosis, routine use of, 112  
   dose of, 289, 290  
   effect on electrocardiogram, 285  
   on vital capacity of lung, 286  
   emboli and, 286-288  
   in extrasystoles, 291  
   harmful effects of, 287  
   inhibition of heart caused by, 93  
   maintenance dose of, 290



- Quinidine in mitral stenosis and auricular fibrillation, 289  
 in non-valvular fibrillators, 289  
 in paroxysmal auricular fibrillation, 199-201  
   flutter, 197  
   tachycardia, (Fig. 24) 193, 314, 318, 319  
 in post-subtotal thyroidectomy for hyperthyroidism, 288  
 for premature ventricular beats, 345  
   ventricular tachycardia, (Figs. 59, 132) 202, 203, 291, 349, 410  
 in prevention of auricular fibrillation, 290  
 respiratory failure and, 287  
   stimulation for toxic action of, 287  
 sudden death and, 93  
 for thyrotoxic heart disease, 148  
 time of action of, 285  
 toxic symptoms from, 289  
 for ventricular fibrillation, 207, 353  
   tachycardia, 121
- RARE forms of heart disease, 137-142
- Reflexes from heart, hypertension and, 127
- Relative insufficiency of valves, in hypertension, 134
- Renal embolism, in coronary thrombosis, 113  
   "surgical abdomen" confused with, 235  
   ischemia (Goldblatt mechanism), hypertension and, 128
- Resection of pericardium, for constrictive pericarditis, 71, 72
- Respiratory failure, quinidine and, 287  
   infections, rheumatic fever and, 20
- Rest, in coronary thrombosis, 123, 124
- Retarded growth, in congenital heart disease, 173
- Retinal changes in hypertension, 129  
   pulsations in aortic insufficiency, 46
- Rheumatic carditis, acute, 26  
   aortic diastolic murmur in, 30  
   apical diastolic murmur in, 30  
   electrocardiographic changes in, 28  
   enlargement of heart in, 29  
   evidence of, 27  
   gallop rhythm in, 28  
   heart block in, 28  
   mitral stenosis confused with, 30  
   pericardial friction in, 30  
   systolic murmur in, 27
- fever, 6  
   abdominal pain and tenderness in, 11  
   symptoms in, 28  
   allergic nature of, 7  
   anemia in, 14  
   antistreptolysins in, 15  
   appendicitis (acute) confused with, (Fig. 69) 11, 28, 29, 235, 239, 357  
   atypical forms of, 8, 10
- Rheumatic fever, bed rest in, 17, 18  
   cardiac involvement in, 13  
   climate in prophylaxis of, 21  
   clinical features of, 14  
   "cold" vaccines in prophylaxis of, 21  
   constitutional factor in, 11, 12  
   diagnosis of typical case of, 15  
   diet in, 17, 19  
   differences in the host in, 9  
   electrocardiographic changes in, 15  
   endocrine glands and, 25  
   epistaxis in, 10  
   exposure to wetness and chilliness and, 20  
   familial factor in, 11  
   "focus of infection" in, 7  
   heart block in, 358, 359  
     (first degree) in, (Figs. 66, 68) 355, 356  
   hyperactive heart sounds in, 9, 14  
   hypertension and, 129  
   infantile paralysis compared to, 13  
   infected teeth and, 20  
   infectious arthritis confused with, 15  
   intravenous salicylate therapy in, 16  
   joints in, 14  
   leukocytosis in, 15  
   lupus erythematosus disseminatus confused with, 15, 16  
   nodules in, 15  
   paroxysmal auricular fibrillation and, 198  
   precordial pain in, 14  
   prevention of recurrences of, by sulfa drugs, 19  
   prognosis of, 22, 23  
   prolonged fever in, 14  
   prophylaxis in, 18  
   public health point of view of, 22  
   rapid heart in, 14  
   regional factor in, 12  
   salicylates in, 16  
   scarlet fever in relation to, 7, 160  
   sclerae in, 12  
   seasonal factor in, 12, 25  
   sedimentation rate in, 15  
   similarity to syphilis, 9  
   skin rash in, 15  
   streptococcus and, 7  
     vaccine therapy in, 16  
   "surgical abdomen" confused with, 234  
   susceptibility to, in relation to angina in parents, 11  
   sweats in, 15, 17  
   tonsils and, 18  
   treatment of, 16  
   tuberculosis simulating, 9  
   typical attack of, 8  
   upper respiratory infections and, 20  
   vomiting spells in, 10



- Rheumatic fever, x-ray treatment of throat as prophylaxis of, 21  
 state, in relation to bacterial endocarditis, subacute, 165, 166  
 valvular disease, bacterial endocarditis subacute, and, 159, 160  
 congenital heart disease and, 173  
 heart block (first degree) in, (Fig. 67) 356
- Right aortic arch, x-ray of esophagus in, 179  
 ventricular heart failure, 253, 255
- Roger's disease. See *Patent ventricular septum*.
- Rupture of aorta, aortic insufficiency in, 118  
 of ventricle, in bacterial endocarditis, subacute, 163  
 causing sudden death, 92  
 in coronary thrombosis, 108, 113  
 of ventricular septum, in bacterial endocarditis, subacute, 163
- Ruptured chordae tendineae, 138  
 valve, 138  
 bacterial endocarditis and, 138, 163  
 diastolic murmur in, 138  
 loud systolic murmur in, 138  
 physical strain and, 215  
 spontaneous, 138  
 trauma and, 138
- SACCHARIN, for velocity of blood flow determination, 258
- Salicylates in rheumatic fever, 16
- Salmon-colored face, in thyrotoxic heart disease, 144
- Salyrgan, for congestive heart failure, 278  
 -theophyllin orally ("S-T-O"), 278
- Scarlet fever, bacterial endocarditis, subacute, and, 160  
 rheumatic fever in relation to, 7, 160
- Sclerae, in rheumatic fever, 12
- Scleroderma heart, 139, 140  
 auricular fibrillation in, 140  
 electrocardiogram in, 140  
 gallop rhythm in, 140
- Seasonal factor, in rheumatic fever, 12, 25
- Sedatives, in congestive heart failure, 270
- Sedimentation rate, in coronary thrombosis, 110  
 in rheumatic fever, 15
- Septicemia, dyspnea in, 225
- Sex, angina pectoris and, 77, 79
- Sexual intercourse, angina pectoris and, 85
- Shock, 252, 253. See also *Peripheral circulatory failure*.  
 causing coronary thrombosis, 106  
 in coronary thrombosis, 109  
 in pulmonary embolism, 211
- "Sighing breathing," 184  
 dyspnea caused by, 223
- "Simple cold," bacterial endocarditis, subacute, and, 161
- Sino-auricular block, (Figs. 64, 65) 354, 355  
 syncope in, 354  
 without heart disease, (Fig. 64) 354  
 node, 297
- Sinus arrhythmia, 303, 304  
 pauses, in aortic stenosis, (Fig. 11) 305  
 carotid sinus reflex and, 305  
 syncope in, 305
- Skin discoloration, in tricuspid stenosis, 54  
 rash, in rheumatic fever, 15
- Sodium cyanide, for velocity of blood flow determination, 258
- Soldier's heart. See *Functional heart disease*.
- Sore throat, bacterial endocarditis, subacute, and, 161
- Spasm of coronary arteries, in angina pectoris, 83
- Splenic embolism, in coronary thrombosis, 113  
 "surgical abdomen" confused with, 235  
 enlargement, in bacterial endocarditis, subacute, 161
- Splinter hemorrhages, in bacterial endocarditis, subacute, 162
- Spontaneous, interstitial emphysema of lungs, coronary thrombosis confused with, 116, 117
- Southey tubes, for congestive heart failure, 281
- "Status epilepticus" in Adams-Stokes disease, 206
- "Steering wheel" accidents, 215
- Strain (physical or mental), cardiac arrhythmias from, 215
- Streptococcus, rheumatic fever and, 7  
 skin test, in bacterial endocarditis, subacute, 165  
 vaccine therapy, in rheumatic fever, 16  
 viridans, bacterial endocarditis, subacute, and, 160
- Strophanthin, 273
- Strychnine, 277
- Stunted growth, in patent ductus arteriosus, 178
- St. Vitus's dance. See *Chorea*.
- Subacute bacterial endocarditis. See *Bacterial endocarditis, subacute*.
- Subdeltoid bursitis, angina pectoris confused with, 91
- Subtotal thyroidectomy, for thyrotoxic heart disease, 147
- Sudden death, 92, 93  
 in angina pectoris, 92, 93, 95  
 in aortic stenosis, 49  
 in complete heart block, 92  
 in coronary thrombosis, 113  
 inhibition of heart causing, 92, 93  
 quinidine and, 93  
 in rupture of ventricle, 92  
 in ventricular fibrillation, 93



- Sulfa drugs, for bacterial endocarditis, acute, 158  
 subacute, 167, 168  
 prevention of recurrences of rheumatic fever by, 19
- "Surgical abdomen," auricular fibrillation confused with, 235  
 coronary thrombosis confused with, 115, 235  
 pericarditis (acute) confused with, 234  
 renal embolism confused with, 235  
 rheumatic fever confused with, 234  
 splenic embolism confused with, 235
- conditions, cardiac conditions confused with, 234-236  
 division of patent ductus arteriosus, 178  
 methods for treatment of congestive heart failure, 291-294  
 operations, anesthetics in, 238  
 for angina pectoris, 102  
 in auricular fibrillation, 237  
 for carotid sinus sensitivity, 208  
 cause of death in, 238  
 in chronic nephritis, 237  
 in congestive heart failure, 237  
 in coronary artery disease, 237  
 coronary thrombosis from, 238  
 for coronary thrombosis, 123  
 for hypertension, 136, 293  
 in hypertension, 237  
 "inevitable" deaths from, 237  
 mortality statistics in, 237  
 peripheral circulatory failure in, 252  
 prognosis of cardiac state and, 236  
 risk in cardiacs of, 234-239  
 "unexpected" deaths from, 237  
 in valvular disease, 237
- Sweats, in coronary thrombosis, 109  
 in rheumatic fever, 15, 17
- Sympathectomy (dorsolumbar), for hypertensive heart failure, 136, 293
- Syncope, in Adams-Stokes disease, 206  
 in aortic stenosis, (Fig. 11) 50, 205, 305  
 atropine for, 205  
 benign, 205  
 in carotid sinus sensitivity, (Figs. 12, 13) 207, 305, 306  
 in cerebral accidents, 208  
 in coronary thrombosis, 110  
 ephedrine sulfate for, 205  
 in epilepsy, 206  
 in heart block (second degree) (Fig. 74) 361  
 (third degree), (Fig. 77) 363, 365  
 in massive hemorrhage, 206  
 in paroxysmal rapid heart action, 190, 205  
 in sino-auricular block, 354  
 in sinus pauses, 305  
 vagovagal reflex and, 305  
 in ventricular fibrillation, (Fig. 61) 207, 351, 353
- Syphilis, angina pectoris and, 79, 80  
 of brain, cerebral hemorrhage confused with, 208  
 malignant arteriosclerosis and, 152  
 rheumatic fever resembling, 9
- Syphilitic aneurysm of aorta, 153  
 aphonia in, 153  
 atelectasis of lung in, 153  
 brassy cough in, 153  
 coronary thrombosis confused with, 118  
 erosion of bone in, 153  
 heart failure and, 153  
 inequality of pupils and pulses, 153  
 localized pulsations in, 153  
 tracheal tug in, 153  
 wiring for, 156  
 x-ray in, 153
- aortic insufficiency, 154, 155  
 angina pectoris and, 80  
 Austin Flint murmur in, 154  
 bacterial endocarditis, subacute, and 160  
 diagnosis of, 154
- aortitis, 152-154  
 angina and, 80, 154  
 aortic second sound in, 152, 153  
 coronary ostia occlusion and, 154  
 Thebesian vessels and, 154  
 Wassermann test in, 153
- heart disease, 152-156  
 treatment of, 155, 156
- myocarditis, 155
- Systolic murmur in anemia, 229  
 in aortic insufficiency, 44, 45  
 "aortic roughening" and, 233  
 in aortic stenosis, 51, 52  
 benign, 181, 231  
 in calcified annulus fibrosus, 227  
 cardiorespiratory, 228  
 classification of intensity of, 229  
 definition of, 228  
 exercise and, 230  
 fever causing, (Fig. 157) 229, 437  
 functional, 228  
 in functional heart disease, 180, 181  
 in hypertension, 229  
 in hyperthyroidism, 229  
 in mitral insufficiency (organic), 226-228  
 (relative), 226-228  
 stenosis, 40  
 in nervous excitement, 229  
 in normal individuals, 229  
 in potential hypertension, 233  
 in rheumatic carditis (acute), 27  
 in ruptured valve, 138  
 significance of, 225-234  
 in tachycardia, 229  
 transmission of, 233  
 in tricuspid insufficiency, 54  
 velocity of blood flow and, 229, 230
- thrill, in aortic stenosis, 50, 51



- TAPPING of pericardial effusion, 66-68
- Tawara node, 297
- Tear of aorta (incomplete), 118
- Teeth, extraction of, bacterial endocarditis and, 170  
infected, rheumatic fever and, 20
- Testosterone, in angina pectoris, 101
- Tetany, from overventilation, 184, 223
- Tetralogy of Fallot, 176, 177
- Thebesian vessels, compensatory coronary circulation and, 154  
syphilitic aortitis and, 154
- Theobrominal, in angina pectoris, 101
- Theobromine sodium salicylate (diuretin), for congestive heart failure, 277
- Theocalcin, in angina pectoris, 101
- Theominal, in angina pectoris, 101
- Theophyllin (theocin), for congestive heart failure, 277
- Thesodate, in angina pectoris, 101
- Thiouracil, for hyperthyroidism, 151
- Thoracentesis, for congestive heart failure, 281, 282
- Thoracic tumor, angina pectoris confused with, 91
- Thrill, diastolic, in mitral stenosis, 37, 38  
systolic, in aortic stenosis, 50, 51
- Thyroid extract, for Adams-Stokes disease, 207  
for myxedema heart, 137
- Thyroidectomy (total), for angina pectoris, 104  
for congestive heart failure, 292, 293
- Thyrototoxic heart disease, 142-152  
apex impulse and, 145  
auricular fibrillation in, 143, 145  
basal metabolism and, 146, 147  
blood pressure in, 146  
capillary pulse in, 146  
cholesterol content of blood and, 146  
Corrigan pulse in, 146  
diarrhea in, 143  
digitalis effect on auricular fibrillation in, 145  
dilatation of left auricle in, 145  
Duroziez's sign in, 146  
glycosuria in, 143  
gray hair in, 144  
hyperactive heart sounds in, 145  
mitral stenosis confused with, 145  
murmurs changing postoperatively in, 148  
operative mortality in, 149, 151  
"pistol shot" in, 146  
prognosis in, 149  
salmon-colored face in, 144  
tachycardia and, 146  
treatment of, 147-152  
anesthesia in, 148  
digitalis in, 147  
Lugol's solution in, 147
- Thyrototoxic heart disease, treatment of, quinidine in, 148  
subtotal thyroidectomy in, 147  
thiouracil in, 151  
vitamins in, 144, 148  
x-ray, 151  
velocity of blood flow in, 146  
weather and, 144  
weight loss in, 143  
x-ray examination for mediastinal gland in, 150  
findings in, 145
- Thyrototoxicosis, angina pectoris and, 77, 81, 82, 85
- Tissue extracts, in angina pectoris, 101
- Tobacco, angina pectoris and, 79, 99  
Buerger's disease and, 99  
premature ventricular beats and, 345
- Tonsillectomy, poliomyelitis and, 19  
rheumatic fever and, 19
- Tonsils, rheumatic fever and, 18, 19
- "Total and permanent disability," heart disease and, 217, 218
- Tourniquets, for congestive heart failure, 281, 284  
for paroxysmal dyspnea, 135, 210
- Toxemia of pregnancy, heart failure in, 139,
- Tracheal tug, in syphilitic aneurysm of aorta, 153
- Trauma, aggravation of pre-existing condition from, 216  
to chest, angina pectoris and, 86  
neurocirculatory asthenia and, 216  
peripheral circulatory failure in, 252  
ruptured valves of heart from 138
- Treatment of heart disease. See *Congestive heart failure, treatment.*
- "Trichterbrust," 225
- Tricuspid insufficiency, liver pulsation in, 53  
54  
pulsating veins in, 53, 54  
relative, 53  
systolic murmur in, 54  
stenosis, 54-56  
age at death in, 55  
ascites in, 54  
auricular (right) dilatation in, 55  
fibrillation in, 55  
blood pressure in, 55  
cervical veins in, 55  
constrictive pericarditis resembling, 55  
71  
duration of symptoms in, 55  
electrocardiogram in, 55  
hypoproteinemia in, 55  
liver enlargement in, 54  
murmurs in, 55  
polycythemia in, 54  
skin discoloration in, 54  
venous pressure in, 55  
valvular disease, 53-56



- Trigeminy, premature ventricular beats and, (Figs. 49, 50) 340
- "Trigger" mechanism, in angina pectoris, 84
- Tuberculosis, bacterial endocarditis, sub-acute, confused with, 164
- functional heart disease confused with, 184
- of lungs, dyspnea in, 225
- mitral stenosis confused with, 38
- rheumatic fever confused with, 9
- Tumors of heart, 140
- auricular fibrillation in, 140
- hemorrhagic pericardial fluid in, 140
- myxoma of auricle, 140
- Typhoid vaccine, for bacterial endocarditis, subacute, 168
- UNDERNUTRITION, bradycardia in, 303
- Urea, in treatment of congestive heart failure, 279
- Uremia, electrocardiogram in, (Fig. 124) 404, 405
- Urginin, 276
- Urinary findings, in coronary thrombosis, 111
- VACCINES, in prophylaxis of rheumatic fever, 21
- Vagal stimulation, as diagnostic aid in paroxysmal rapid heart action, 203, 204
- in paroxysmal rapid heart action, 203, 204
- Vagovagal reflex, syncope and, 305
- Valsalva's experiment, in treatment of paroxysmal auricular tachycardia, 192, 193
- Valvular disease, pericarditis, chronic, non-constrictive, confused with, 75
- surgical operations in, 237
- Valvulotomy, for mitral stenosis, 291
- Vascular vulnerability, and hypertension, 128
- mitral stenosis and, 42
- Velocity of blood flow, in acute pulmonary edema, 259
- anemia and, 146
- in aneurysm of aorta, 259
- in cancer of lungs, 259
- in congestive heart failure, 258
- in emphysema, 259
- fever and, 146
- hyperthyroidism and, 146
- measurements of, 258, 259
- in pneumonia, 259
- systolic murmur and, 229, 230
- Venous pressure, in congestive heart failure, 254, 256, 259
- in pericarditis, constrictive, 68, 70
- in tricuspid stenosis, 55
- Ventricular alternation, sounds in, (Fig. 151) 429, 430
- aneurysm, diagnosis of, 113, 114
- Ventricular fibrillation, in Adams-Stokes disease, (Fig. 62) 352
- adrenalin and, 351
- angina pectoris and, (Fig. 63) 353
- coronary artery ligation and, 351
- in coronary thrombosis, 113
- quinidine for, 207, 353
- sudden death caused by, 93
- syncope in, (Fig. 61) 207, 351, 353
- "flutter," (Fig. 60) 350, 351
- hypertrophy (right), from prolonged left ventricular failure, 375, 376
- mural thrombosis, in coronary thrombosis, 108
- preponderance. See *Electrocardiogram, axis deviation.*
- septal defect, bacterial endocarditis, sub-acute, and, 160, 163
- Vital capacity of lungs, age and, 89
- in angina pectoris, 89
- in congestive heart failure, 256, 259
- 260
- general considerations of, 88, 89
- obesity and, 88, 89
- in pregnancy, 242
- quinidine, effect on, 286
- Vitamin B deficiency, beri-beri heart from, 139
- Vitamins, for congestive heart failure, 271
- for thyrotoxic heart disease, 148
- Vocal cord paralysis, atrial septal defect and, 175
- Vomiting for paroxysmal auricular tachycardia, 192
- in rheumatic fever, 10
- WASSERMANN test, in syphilitic aortitis, 153
- Weakness, in coronary thrombosis, 109
- Weather, thyrotoxic heart disease and, 144
- Weight, difference between "normal" and "optimum," 20
- Wenckebach phenomenon, in heart block, (Fig. 71) 359, 361
- Wilson's central terminal for electrocardiography, 416
- Wiring, for syphilitic aneurysm of aorta, 156
- Wolff-Parkinson-White syndrome, (Figs. 131, 132) 408-411
- XANTHOMATOSIS, angina pectoris and, 80
- x-Ray, in aortic stenosis, 53
- in calcified aortic stenosis, 51
- in coarctation of aorta, 174
- in congestive heart failure, 264
- examination for mediastinal goiter, for thyrotoxic heart disease, 150
- in mitral stenosis, 39
- in patent ductus arteriosus, 177, 178
- in pericardial effusion, 66
- in pericarditis, constrictive, 70, 71



- |  |  |
|--|--|
| <p>x-Ray in right aortic arch, 179<br/> in syphilitic aneurysm of aorta, 153<br/> in thyrotoxic heart disease, 145<br/> treatment, of adrenals for angina, 101<br/> for lupus erythematosus disseminatus,<br/> 141</p> | <p>x-Ray treatment of throat as prophylaxis of<br/> rheumatic fever, 21<br/> for thyrotoxic heart disease, 151</p> <p>Yellow vision, from digitalis, 276</p> |
|--|--|



ALLAMA IQBAL LIBRARY



4699





